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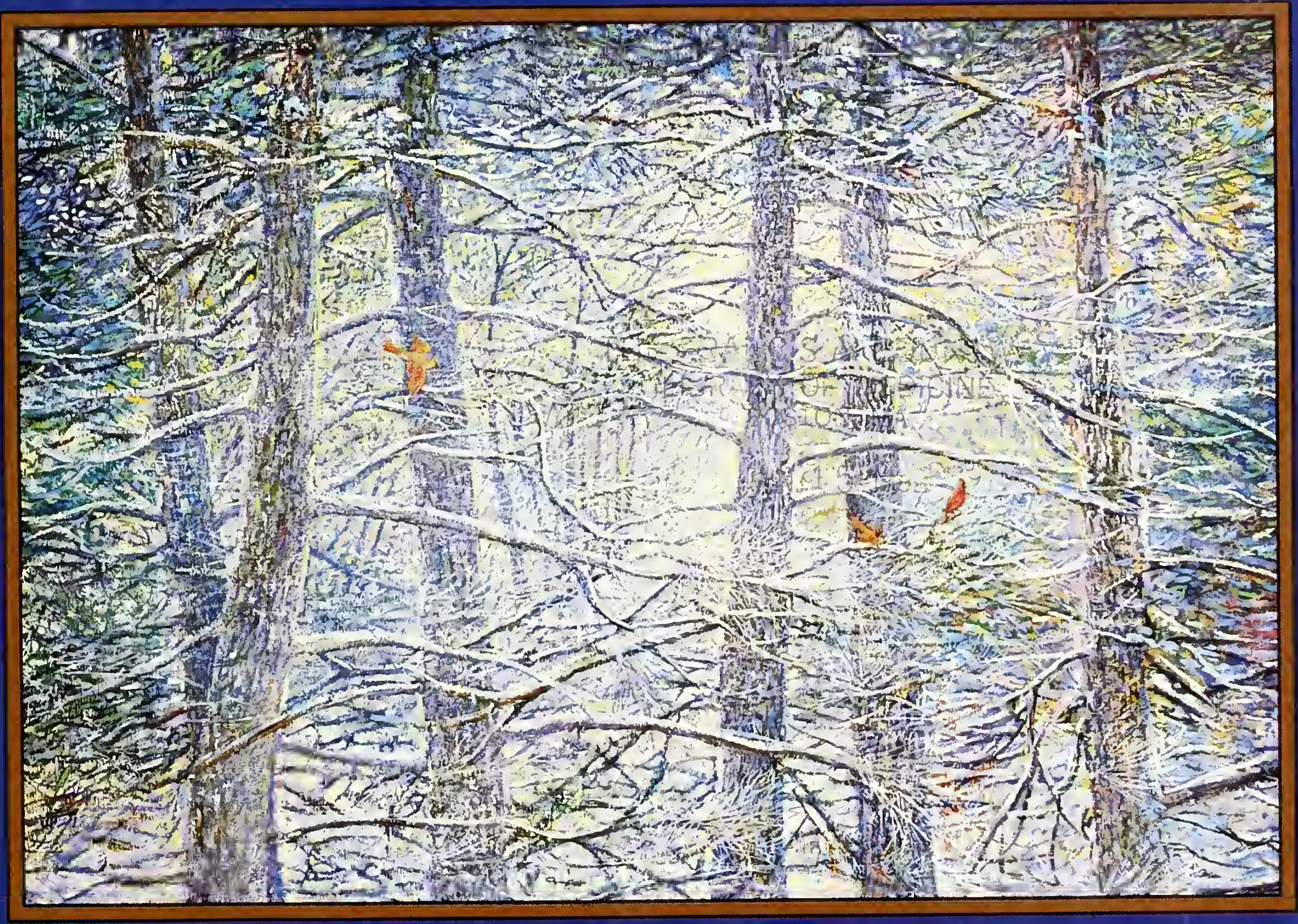
Missouri Medicine

Journal of the Missouri State Medical Association

January 1992



In this issue:

- Bronchopulmonary Lavage for Pulmonary Alveolar Proteinosis
- Massive Gastric Hemorrhage due to Small Vessel Erosion (The Dieulafoy Lesion)
- Racial Differences in Cardiovascular Disease Mortality and Risk Factors

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This month's cover is by Terry L. Martin, artist and art instructor at William Woods College in Fulton, MO. Mr. Martin's fine work has enhanced several past *Missouri Medicine* covers and we welcome this opportunity to feature one of his more recent works.

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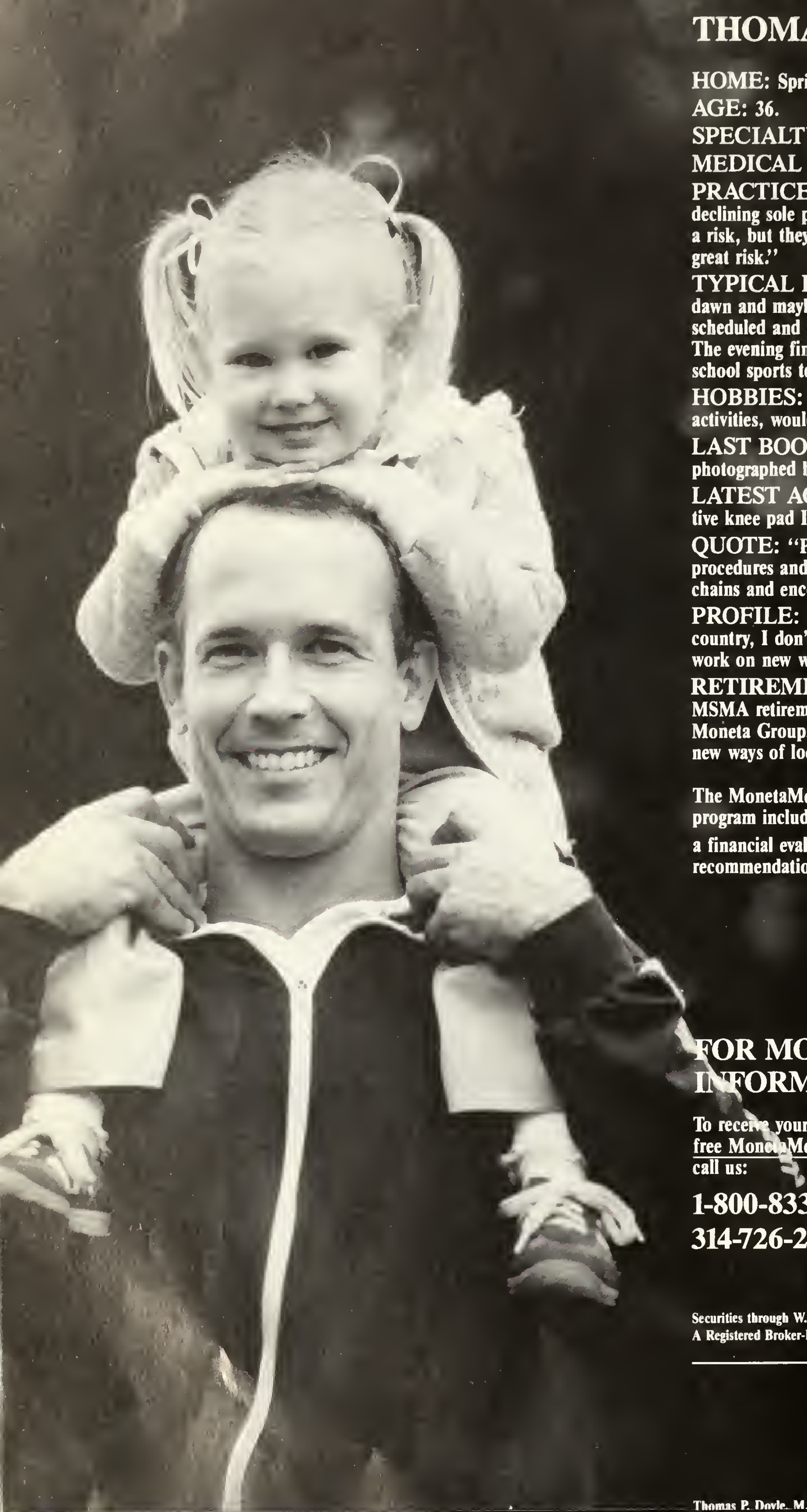

THOMAS P. DOYLE, M.D.

HOME: Springfield, Missouri.

AGE: 36.

SPECIALTY: Orthopedic Surgery.

MEDICAL COLLEGE: University of Missouri, 1978

PRACTICE: "I recently purchased an established but declining sole practitioner office from a retiring physician. It's a risk, but they say that great deeds are usually wrought at great risk."

TYPICAL DAY: "Let the horses out of the corral at dawn and maybe even go for a ride. Then, it's surgery if scheduled and a day of torn ligaments and sprained ankles. The evening finds me on the sidelines of one of our high school sports teams."

HOBBIES: Horseback riding and golf. "If I combined activities, would it be polo?"

LAST BOOK READ: Missouri: Images of Nature, photographed by Charles Gurche.

LATEST ACCOMPLISHMENT: "Testing a protective knee pad I'm developing for use by football players."

QUOTE: "Physicians are being deterred from trying new procedures and therapies in patient care. We need to cut the chains and encourage individual innovation."

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Missouri Medicine

Journal of the Missouri State Medical Association

Vol. 89, No. 1 January 1992

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Printing Office: The Ovid Bell Press, Inc., Fulton, MO 65251
Missouri Medicine (ISSN 0026-6620) is published monthly by the Missouri State Medical Association, P.O. Box 1028, 113 Madison St., Jefferson City, MO 65102.
Subscription rates: $20 per year.
Second class postage paid at Jefferson City, Missouri and at additional mailing offices.
Postmaster: Send address changes to Missouri State Medical Association, c/o Membership Dept., P.O. Box 1028, Jefferson City, MO 65102.

The articles published in *Missouri Medicine* represent the opinions of the authors, and do not necessarily reflect the official policy of the Missouri State Medical Association.
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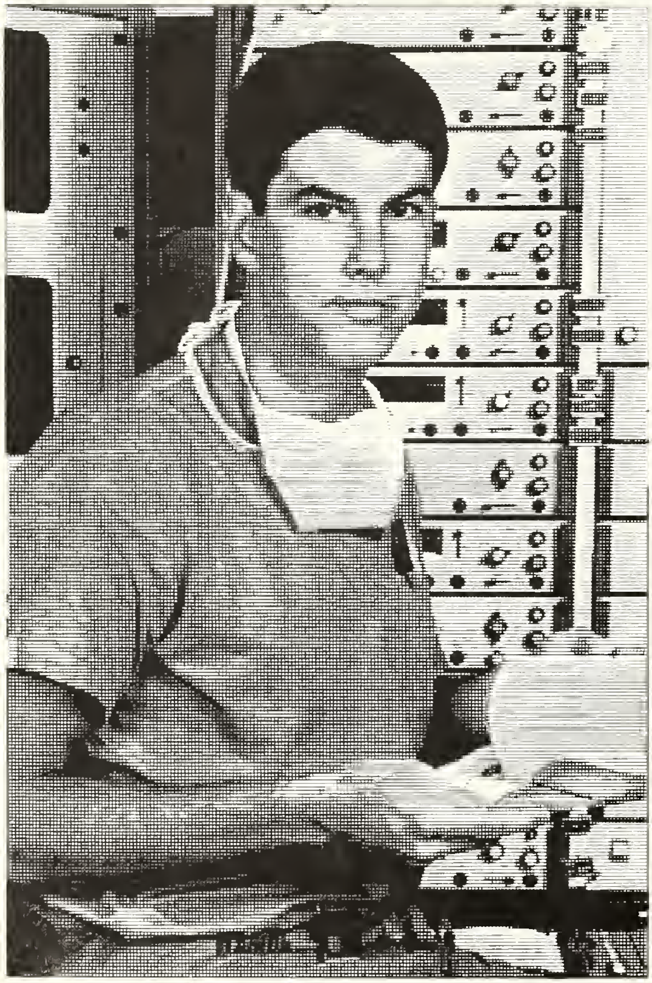

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EDITOR'S DESK

by
Donald G. Sessions, M.D.
Editor

ADVANCE DIRECTIVES: BEGINNING THE PROCESS

She was an elderly 72-year old woman who lived at home with her husband and adult daughter. Prior to admission for treatment of cardiomyopathy she had completed a Living Will. Cardiac surgery was performed and in the post operative period she was placed on the ventilator for what was expected to be a short time. However she became dependent on the ventilator and it was not possible to wean her from the respirator. She was transferred to a specialty floor for ventilator dependent patients.

Family members visited daily and spoke with the nursing staff and social workers frequently about concerns and feelings. At their request a conference was held with her physician who indicated plans to attempt weaning for approximately a month. This course of action was not successful and, as the following weeks turned into months, the family expressed increasing frustration with the patient's lack of progress and intermittent confusion. She was placed on a waiting list for a nursing home as the family felt incapable of managing her care.

As it appeared likely that she would never be able to come off the ventilator, the family expressed guilt about having talked her into the surgery, her poor quality of life, and the mounting medical bills. The daughter and the husband eventually came to the conclusion that the patient would be better off not living, and met with the physician, the nursing staff, and the social worker to discuss removing the ventilator. The physician agreed to ask the patient if she wanted this. The patient did not give an answer either way, and it was uncertain whether she could make this decision with her intermittent confusion. A few more weeks passed without an answer from the patient. The family was increasingly frustrated . . . .

The new patient self-determination act, the Danforth Bill, requires patients in health care facilities receiving Medicare or Medicaid funds to be informed of their right to refuse life-sustaining treatment. It applies to hospitals, hospices, home health agencies, nursing homes, and HMOs. In addition to informing patients about their right to refuse life sustaining care, inpatient facilities must tell them of their right to complete an advanced directive and a medical durable power of attorney. Surveys have indicated that only 4% to 17% of patients have completed a living will or a durable power of attorney.

The new law has the ability to initiate discussion about treatment options for individual patients. This will clearly have patient self-determination and autonomy as its worthy goals. Physicians can facilitate this by beginning discussion before patients get to the hospital. The doctor's office is the ideal place to begin the process. Physicians can now routinely discuss advance directives with the purpose of facilitating patients to guide their own care. A discussion of the patient's values and desired treatment goals can occur before offering available

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Editor's Desk

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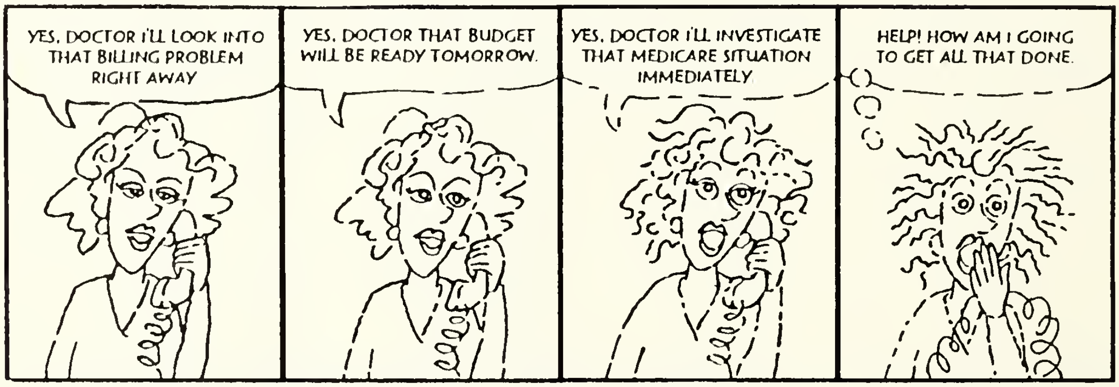
medical technology. This process can be used to evaluate how well patients can make decisions and how realistic their health care goals are. And for those physicians who are not willing to discuss these deeply troubling issues of withdrawal of treatment with healthy patients, it can now be done under the guise that we are "required" to address these issues by the government.

The patient self-determination act will definitely nudge both patients and physicians to talk about topics that have been less than comfortable to think about. Few surgeons, for instance, are at ease in discussing the right to refuse life-sustaining care with healthy young patients who are about to undergo an elective procedure. Yet this is just the patient who is most at risk if some catastrophe occurs. Few of use would have felt comfortable discussing this with patients in the age group of Nancy Cruzan or Christine Busalacchi.

In looking at our ventilator-dependent patient's circumstances it would be interesting to surmise what might have happened if the same situation had occurred several years from now. Since it is fairly common for patients undergoing cardiac surgery to require ventilatory support in the immediate post-operative period, then the possibility of her becoming ventilator dependent might well be addressed in the precess of informed consent. If that real possibility had been discussed up front with the patient she then could have made her wishes known with certainty and could have named a health care surrogate to facilitate those wishes occurring. The problem of competency will always be difficult both for the physician and the family. Beyond the question of legal competency it is often not completely clear whether a person is fully competent to understand a complex medical situation and further, whether that person is capable of making an informed choice. By focusing on these issues before they occur the patient self-determination act can help patients participate in their own care in a way that is consistent with their personal values and desires. That this can allow physicians to care for patients in a space of increased certainty should be assuring to us all.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THE PRESIDENT'S PAGE

by
Dorothy D. Reister, M.D.
Missouri State Medical
Association President

HERE COMES 1992

This is and has been a time of celebrations Thanksgiving, Hanukkah, Christmas and the New Year. Each of these has its own traditions and in addition each family has its own special observances. Also it is a time of giving and helping the less fortunate. It's a time at the end of the year when bad debts are written off . . . essential donated care. (Remember the program that Dr. Gary Dyer started and be sure to report that information to the Association office.) It is a time of year-end donations to church, charities, college, medical school and don't forget AMA-ERF, requesting that it be credited to Missouri. In addition there are the social events which give us opportunities to gather at times other than "meetings".

So what do we see as the god Janus turns his head to the New Year? For the Missouri State Medical Association there is political action. The Legislative Commission chaired by Dr. Jerry Murrell of Columbia met in November and reviewed and discussed up-coming legislation. We desperately need to improve our relationships with the legislators; we can have more and more active Key Contact people. How about making contacts while legislators are home for the holidays? Let them know that you are an *informed* source of information . . . not just more of what they read in the newspapers. There will be bills on bicycle safety, fireworks, midwifery, equality in Workers Compensation, Medicaid reimbursement, universal reporting insurance form, universal coding (bring medicaid coding into the system used by everyone else) and many others. We really need to plan a real "March on the Capitol".

Another area of activity is increasing the cooperation between the Missouri Osteopathic Association and the Missouri State Medical Association. The executive committees of the two organizations have met and established some goals. Together we will work for a more comprehensive definition of the practice of medicine. Medical legislation has a much better chance of passage if it is backed by ALL of organized medicine. Both groups have the same problems with Medicaid and we perhaps will be joining forces to pursue a judicial route as the hospitals and nursing homes have done (and won). Both groups have similar problems with the

Continued on page 10

A POINT OF PERSONAL PRIVILEGE

I am taking this opportunity to express my thanks and unbounded appreciation to all the Missouri State Medical Association members and the staff in Jefferson City and the component societies who have been so thoughtful and generous in their caring and concern in my recent illness. I've been astounded by the out-pouring of flowers, cards and calls. It makes me very humble. I'm continuing my Association activities and your president has already been "on the road again". Thank you all from the bottom of my very grateful heart.

Dorothy D. Reister, M.D.

The President's Page

Continued from page 9

Board of Healing Arts and again a presentation made by ALL of organized medicine will be that much more effective.

Some health care plans exclude osteopaths in blanket fashion without individual evaluation. We should continue to decrease barriers and discuss in depth the items which seem insurmountable. After all, the mountain climb starts with the first step.

Another mountain is the Board of Healing Arts fees and tactics. The Board is the source of most of the complaints I hear around the state. Physicians feel the dues increase is too steep, that the investigating officer's tactics upon arrival at a physicians office are still rather Gestapo-like, that there is far too long a delay from investigation to letter of disposition of the matter, in which time the physician's anxiety is ignored. Prompt response is a judicial right!

Also, a risk management program that physicians can participate in should be given cooperation and CME credit. If the Board of Healing Arts truly is interested in improving the practice of medicine in the state then they should be encouraging both risk management programs and prompt reporting back to physicians.

Here we come 1992 ... hoping to be bigger, stronger, more active, yet fighting for universal access to health care in the American tradition. We know the Canadians, British, others come to our country for health care BECAUSE WE ARE THE BEST

Answers to the last puzzles:
The five star generals were:
Douglas MacArthur,
Dwight Eisenhower,
Omar Bradley,
George Marshall and
Henry (Hap) Arnold

The person was fired from the
M & M factory because:
She was throwing out the W's.

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AUXILIARY MESSAGE

by Mary Hunkeler,
MSMAA President

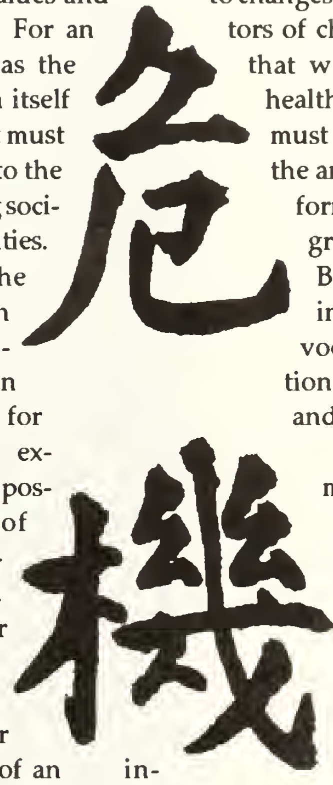
Change Can Bring Us Risk and Opportunity

January represents not only a change in the calendar year, but a certainty that change will occur at an ever increasing rate in our society today. In *Future Shock*, Alvin Tofler writes of the difficulty of our adjusting to "the roaring current of change, a current so powerful today that it overturns institutions, shifts our values and shrivels our roots." For an organization, such as the Auxiliary, to sustain itself and remain viable, it must find ways to adjust to the rapid changes facing society and our communities.

By combining the characters that mean "danger" and "hidden opportunity" in their written word for "crisis, the Chinese express not only the possible outcomes of change, but a philosophy of change. Change is never easy. Usually, there is an element of crisis in any major change, in the life of an individual or an organization. But, if we look at both the "dangers" and the "hidden opportunities" inherent in crisis we realize that change and adjustment to change become positive instead of negative. The challenge of a possible name change for the AMA Auxiliary (and for us) is both a risk and an opportunity.

The Auxiliary is not an isolated organization; changes in our society and communities affect us. How we respond to those changes is critical. We must not be afraid of change, because we see only the dangers. We must see the opportunities presented to us to respond appropriately to our communities and the Auxiliary's role in that community. The Auxiliary should not just react to changes. We should also be initiators of change, especially change that will affect the quality of health in our communities. We must be willing to take risks in the area of AIDS education, to form coalitions with other groups as in the Shaken Baby Syndrome project, to initiate new projects, to advocate for medical legislation and to educate for health and wellness.

As an Auxiliary, we must always focus on the opportunities presented to us in any change situation whether it is the way we respond to changes or the way we initiate changes in our communities. Or in a name change. As the new year progresses and we confront change, let's remember the Chinese characters for crisis and seek out the "hidden opportunities". ■



Editor's Note: The word "Crisis" , above, written in Chinese characters, was done by Kenneth K.C. Siu, M.D., retired general surgeon, Jefferson City, Missouri

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PRACTICE MANAGEMENT

by Janice K. Woerth, Ph.D.

Do you have a question for Dr. Woerth? Include your questions along with your name, address and phone number and send them to:

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Or call her direct:
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Although your name will not be used in *Missouri Medicine*, please include your name and phone number so that Dr. Woerth may contact you if she needs additional information to answer your question.

Dear Dr. Woerth,

I read in your Practice Management column that the CPT codes are changing. Does this mean that the codes will have different numbers? If so, won't I have to change my superbill?

C.F.

Dear C.F.,

The new CPT Codes for evaluation and management, i.e., office, hospital, nursing home, emergency room, home, case management, preventive medicine, and critical services, that will be published in the 1992 CPT book do have new numbers. The codes for consultations will also have new numbers. All these codes will now be in the 99,000 series. In other words, each code will begin with numbers 99 followed by three other numbers. For example, the office and other outpatient services codes will range from 99201-99205 for new patients and 99211-99215 for established patients.

Because of these numerical changes in the codes, it will be necessary to update your superbill (encounter form or fee ticket). You will also need to change these codes in your computer. Be aware that more than the numbers of these codes are changing. The AMA has emphasized that the definitions are entirely different from the previous codes we have been using for evaluation and management services. The AMA has also emphasized that there is no crossover from the previous codes. This means that the 1991 definition of a brief office service is not the same as the 1992 Level 1 or 2 (or any other level) office service. Thus, it will be necessary to learn the new definition of these evaluation and management codes in order to appropriately use them.

It was concluded from the field studies conducted by the AMA that "the CPT Editorial Panel's proposed revisions to visit and consultation codes, provided substantial evidence that the new evaluation and management codes can be easily and reliably used by physician". So while we are faced with some changes in the way medical services are reported, these code changes should simplify the reporting as well as improve it's accuracy.

Dear Dr. Woerth,

One of our patients unfortunately has been found to be HIV positive. When we tried to assign the ICD-9-CM code for this diagnosis, we find the ICD-9-CM very confusing. Can you explain how it is used?

Dr. L.F.

Dear Dr. L.F.,

To code for the Human Immunodeficiency Virus (HIV), you must start in Volume 2 of the ICD-9-CM under the heading Human Immunodeficiency Virus. There you should find a chart that lists, on the left, HIV related diseases and, on the right, six columns indicating the conditions of the HIV disease. These codes were added to ICD-9-CM in October of 1986. So if you cannot find this chart in Volume 2, you need to update your ICD-90-CM books.

HIV associated conditions are represented in the columns as follows:

042 -HIV infection with specified secondary conditions or malignant neoplasms or AIDS

043 -HIV infection causing other specified conditions, including AIDS-like syndrome, AIDS-related complex, or ARC

044 -other HIV infections not listed above

All HIV conditions being treated at the visit need to be included on the claim form as well as one code from the 042-044 series. You code the most advanced stage of the HIV infection that the patient is in. In other words, category 042 takes priority over 043 and 044; 043 takes priority over 044.

Notice on the chart that each of the three stages of the HIV infection is listed in the columns with the word(s) "with" or "due to", i.e., with HIV or due to HIV. The "with" columns for the three stages of the disease indicate that the patient's condition is association with that stage of the disease. The "due to" columns for the three stages of the disease indicate that the patient's condition is due to that stage of the disease.

It is obvious from the information above that coding for HIV can be complex. Remember that the code(s) you find on the HIV chart in Volume 2 of ICD-9-CM must be looked-up and verified in ICD-9-CM Volume 1.

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Letters to the Editor

Dear Dr. Sessions:

I completed a grant training program this year, and through *Missouri Medicine* I want to tell other Missouri physicians so they also can take advantage of it.

The small number of lipid specialists in the country, localized to university medical centers and large cities, cannot meet the growing demand for consultative services from primary care physicians when they encounter patients with complex lipoprotein disorders.

The Lipid Disorder Training Course sponsored by the American Heart Association and Bristol-Myers Squibb intensively trains intermediate-level clinical lipid specialists over a 3-day period in six centers. These physicians then provide consultation and establish standards of practice for the treatment of these disorders at a local level.

University lipid research-teaching centers across the country vied for the opportunity to be one of the six training centers for the program. The Lipid Research Center at Washington University in St. Louis was chosen to administer the program for physicians in eleven states including Missouri. Drs. Gus Schonfeld and Anne Goldberg directed the program and contributed much to the very large syllabus provided for the course.

The program lasted 3 days, cost only $60, and gave 23 hours of CME. Most of the teaching was didactic but there also was a 2 hour hands-on session in the clinic, e.g. palpating xanthomata, a 2-hour excellent session with the research dieticians, a 2-hour session on strengths and limitations of laboratory measurements of lipoproteins, and an outstanding interview session with a group of unusual patients with complicated treatment regimens.

Thirty-six up to date slides were provided for my use after the course. I have found them invaluable in the six talks that I have given to physicians in the West Plains area. These talks have "spread the word" about diagnosis and treatment of lipid disorders.

A personal aside. The first day of the course, we reviewed our own three-day diet history and had blood drawn. I was astounded to find I had the highest total cholesterol in the group, 288 mg./dl. I immediately used the information I learned at the course, especially from the research dieticians. I was very pleased to see how effective diet alone can be. In three months my total cholesterol was, and has remained, less than 160 mg./dl.

I hope all physicians in Missouri who have a bent in this direction can take advantage of this unusual opportunity for the benefit of themselves, as well as all of the patients who urgently need diagnosis and therapy of their lipid disorders. The course has expanded my practice in that area dramatically.

Harold V. Werner, M.D.
West Plains

Dear Dr. Sessions:

Dr. Wilfred Wooldridge's editorial "Geriatric Suicide" in *Missouri Medicine*, November, 1991 is timely. Suicide rates have increased alarmingly, especially among the elderly and among teens. What's the answer to the problem of what I would call "existential depression" and increasing suicides? Dr. Wooldridge says probably the answer is not medical but sociological.

I have practiced psychiatry for many years and am now semi-retired. I would like to express another opinion: I don't know of any social welfare program which has significantly improved people's sense of worth and meaning for their lives. I have to agree with the opinion an internist recently expressed: "Most mental and emotional illnesses are but symptoms of an underlying spiritual problem."

As I see it, America and the medical profession, including psychiatry, have on the whole turned away from the spiritual values which have been so vital to our well being -- for both individuals and society. I do not make this statement lightly but from personal experience.

Early in my career in psychiatry I had many years of psychoanalytic therapy for both training purposes and personal problems like shyness. I was benefited by my therapy, yet I accomplished much less than what I had hoped. I was dissatisfied and felt deeply that something was missing. But in 1981 I had a spiritual experience that dramatically transformed my beliefs and values. I had been literally a "doubting Thomas" from Missouri and had gone some 20 years without any religious activity. Once I even said to a colleague, "We don't need God, and certainly He doesn't need us." But I thank God that He made me realize how wrong I was! A relationship with Him is what I needed most of all! I turned to Him in sincere repentance and I came to have the most marvelous experience of my entire life! I felt the Spirit of God in a way I never even imagined! A wonderful warm feeling flowed into my heart and throughout my body. I felt love, joy and peace as I never had before. Words can't possibly convey that joyous experience. Now I knew with full assurance in my heart that God is real! He

Continued on page 16

continued from page 15

loves us as no one else can. And His word, the Bible, is truth. For the first time ever I felt a real sense of meaning and value for my life.

This experience also changed my body: I rapidly lost 12 pounds of excess weight without a thought of dieting! I lost a craving for sweets. People said I looked 10 years younger. And I felt 10 years younger!

The Bible has several names for this experience with the Spirit of God, including "be partakers of the divine nature". (2 Peter 1:4) Now I know well what Pascal (1623-62) meant when he said, "There is a vacuum in the heart of each one of us the exact size and shape of God, and it will never be satisfied until filled with God." I am much happier and I know my life will never be the same!

I know now that any one who has received God's Spirit into his or her heart will experience real meaning and joy in this life -- and will not lose faith in God and His promises: both in this life and the life to come. I also know that even the most miserable circumstance need not cause loneliness and despair, if one will turn to God in humble and sincere repentance, and then seek God's answer to such a problem. I am also sure that the most scientific, most humane and most highly funded programs of society without God will never satisfy the unfulfilled hearts that need God's Spirit and His love, peace and joy.

Sincerely,
Thomas N. Davis, III, M.D.

Dear Dr. Sessions:

I read with interest the guest editorial by Dr. Wilfred Wooldridge titled "Geriatric Suicide, A Pattern Shift" in the November issue of Missouri Medicine.

I agree with Dr. Wooldridge that Major Depression and the threat of suicide in older adults should never be taken lightly. The two case histories presented by Dr. Wooldridge of an 80 year old farmer and a 72 year old woman who lived alone typified classical Major Depression that is in need of intensive psychiatric care. It was kind and caring of Dr. Wooldridge to take the time to visit one of these patients and to give encouragement and support. It is imperative, however, that we recognize Major Depression in the elderly as a killer disease, with encouragement and support being only instruments to urge such a patient to enter the hospital for effective antidepressant therapy. Electroconvulsive therapy followed by appropriate antidepressant medications, close monitoring and involvement of the family or social agencies in the care of an elderly patient following hospitalization and discharge are the only appropriate and most effective means for avoiding the high risk of suicide. Depression in older adults is an extremely common disease and a frequently discounted and dismissed serious illness.

Marcel T. Saghir, M.D.
St. Louis

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Has The Missouri State Board of Registration For The Healing Arts Gone Too Far?

by Arthur H. Gale, M.D.

The Missouri State Board of Registration for the Healing Arts has proposed increasing the fee for physicians' license renewal from $75.00 to $120.00. License fees were $10.00 per year until 1986, when they increased to $30.00. In 1990, they jumped to the present level of $75.00. The proposed increase would generate more than $880,000 for the State Board.

Is this fee increase justified? Does the State Board of Registration for the Healing Arts need to increase its activities, or do its activities need to be maintained at the present level, or do they need to be scaled back?

Recently a retired physician showed me a letter that he had received from the State Board of Registration for the Healing Arts reprimanding him for improper medical practice. His wife has been hospitalized for a long period of time. At the time of her discharge, her attending physician had prescribed Darvon Compound. The retired physician called in the exact prescription prescribed by his wife's attending doctor to the hospital pharmacy under his own name because he wanted the prescription ready at the time of his wife's discharge and did not want her to wait to have it filled. He was reprimanded by the State Board for writing a prescription for a patient without having access to that patient's records.

Criticisms of over-zealous investigations and charges against physicians were leveled against the State Board of Registration for the Healing Arts at a recent meeting attended by representatives of both the Board of Healing Arts and the Missouri State Medical Association.

Similar charges have been leveled at state licensing boards in other states, most notably Massachusetts. In Massachusetts three nationally known, respected, well intending physicians were sanctioned for minor infractions that they were not even aware of. These physicians were then listed like common criminals in the Massachusetts Medical Association Journal along with those physicians who had committed truly felonious criminal acts like selling controlled drugs to the public. The Massachusetts Medical Society and physicians throughout Massachusetts have been highly critical of the Massachusetts licensing board for their actions.

Why are the state licensing boards increasing their size, employing more investigators, and becoming more zealous in weeding out "bad doctors?"

The answer was given by a representative to the Board of Healing Arts who spoke to the Council of the St. Louis Metropolitan Medical Society about a year ago. He said that if the medical profession does not show that it is trying to discipline itself, then the "Naderites" and consumer organizations will take over that function. The implication was that the "Naderites" and the consumer organizations were forcing the State Board of Registration for the Healing Arts to become more aggressive in ridding the profession of "bad apples."

Let us examine Ralph Nader and his consumer ally for medical affairs, Sidney Wolfe, M.D., and their organizations more closely.

In her recently released book, *Trashing The Planet*, Dixie Lee Ray, a respected scientist, former head of the Atomic Energy Commission, and governor of the State of Washington, describes Ralph Nader and his consumer organization as leaders in the opposition to the use of many safe products of modern technology. She points out that the *consensus of expert scientific opinion* will conclude that a particular product is safe according to the *best available evidence.* But the Naderites will quote dissenting views from scientists who are often out of the "main stream" of scientific thought. The scientists with proven credentials are put in a difficult position. They cannot and do not wish to defend their views against outrageous charges. Therefore, the product is never introduced or removed from the market place. Ray goes on to say that science has its quotas of "charlatans and quacks" just as every profession does. Physicians are well acquainted with this scenario with regard to the testimony of some expert witnesses in malpractice trials.

Nader has made a career of demanding full disclosure from corporate America about all aspects of their activities including lobbying. However, Nader himself has refused to divulge where much of his financial support comes from. An article in the September 17, 1990 issue of *Forbes Magazine* reveals how Nader has opposed all aspects of tort reform, how he receives huge contributions from trial lawyers, and how he has enlisted the support of trial lawyers in his crusades. When he speaks at conventions of trial lawyers he receives not only large honorariums but he is hailed as a conquering hero. Strictly speaking, Nader and his organizations do not truly represent independent "consumer organizations" but just another interest group with its own agenda, Nader's agenda.

Ralph Nader's consumer group, Public Citizen, has only 50,000 members. Even if the membership in these organizations were greater there is no proof that they speak for the majority of the American public. Their memberships are seldom polled, yet, these organizations wield enormous power, testifying before Congress frequently, as if they represented millions of consumers.

More pertinent to the medical community are the actions of Sidney Wolfe, M.D. He has been a staunch critic of just about all aspects of the medical establishment. Wolfe has never practiced medicine. He is not board certified, nor is he a respected scientist with expertise in any particular field.

However, Wolfe passes himself off as an expert on bad drugs and bad doctors. He has written a book on "bad drugs." He has also published a book on "bad doctors" listing those doctors who have been sanctioned by their state licensing boards. Some of these sanctions have been for trivial infractions.

Just writing a book on "bad doctors" is not enough for Wolfe. He believes that state licensing boards are not sanctioning enough physicians. Missouri is at the top of the list for sanctioning doctors. That is good, says Wolfe, but there are many more "bad doctors" out there no being sanctioned.

Is the State Board playing a numbers game to please Wolfe, Nader and their consumer organizations? The State Board ought to know that Wolfe and Nader and their consumer organizations' appetite for "bad doctors" can never be satisfied.

Is it a good thing for Missouri to be at the top of the class for sanctioning physicians? Has the State Board achieved its top ranking by sanctioning conscientious, well intending physicians for nit picking, petty, infractions?

The actions of the Board of Registration and Healing Arts should not be tailored to meet the demands of vociferous, non-representative, self-appointed guardians of the public interest.

The physician members of the Board of Healing Arts should restrain, not encourage, some of the activities of the professional staff. There should be a committee in the Missouri State Medical Association to act as a repository for complaints about the Missouri State Board of Registration for the Healing Arts and act as a liaison between organized medicine and the Board.

Over-regulation is one more hassle which keeps qualified people from entering the medical profession and drives qualified physicians out of the profession, thereby limiting access to care. The Board of Healing Arts does not need to expand and become a more bloated bureaucracy. Fees should not be raised to $120.00

■

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The Missouri State Medical Foundation — MSMF — was created by the Missouri State Medical Association for the express purpose of giving financial assistance to deserving young Missourians whose goal is to be one of tomorrow's physicians. Medicine needs enthusiastic and dedicated doctors to tackle the challenges facing medicine. We need your help to propel our young leaders into the future.

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Missouri Medicine prides itself on its beautiful covers. The journal repeatedly receives compliments on its featured art. You can help to ensure that the tradition of creative covers for *Missouri Medicine* continues. We would love to see your photos/paintings of Missouri, animals, birds, flowers, landscapes — anything with a taste of Missouri. If you have a photograph/painting you would like to see on our cover, we want to see it! Send a 35 mm slide of your work to: MISSOURI MEDICINE, 113 Madison, P.O. Box 1028, Jefferson City, MO 65102. Don't be shy! For questions call Dennis Weiser, Managing Editor at 314/636-5151.

RENEWAL FEE -
THE COST OF YOUR LICENSE AS A PROFESSIONAL

by Frederick A. Tromans,
Executive Director, Missouri Board of Healing Arts

We understand that some physicians are concerned about the increase in the renewal fee. Indeed, considering that the fee has risen from $10 in 1987 to $120 in 1992, concern is justified. Both the $10 fee and the $120, however, are exceptional.

Five years ago, because a balance had accumulated in the Healing Arts Fund, the fee was held to $10 so that none of the money in the fund would be diverted to the state's general fund (the law states that if the fund reaches two times the annual expenditures, any money over that amount is forfeited into general revenue).

Since that time there has been a significant increase in consumer complaints, as shown in Figure 1.

We are required by law to investigate each complaint. Consequently, we requested from the legislature appropriation from the Healing Arts Fund (all spending by the Board must, by law, come from the fund) to add five investigators, two part-time physicians, and support staff for them.

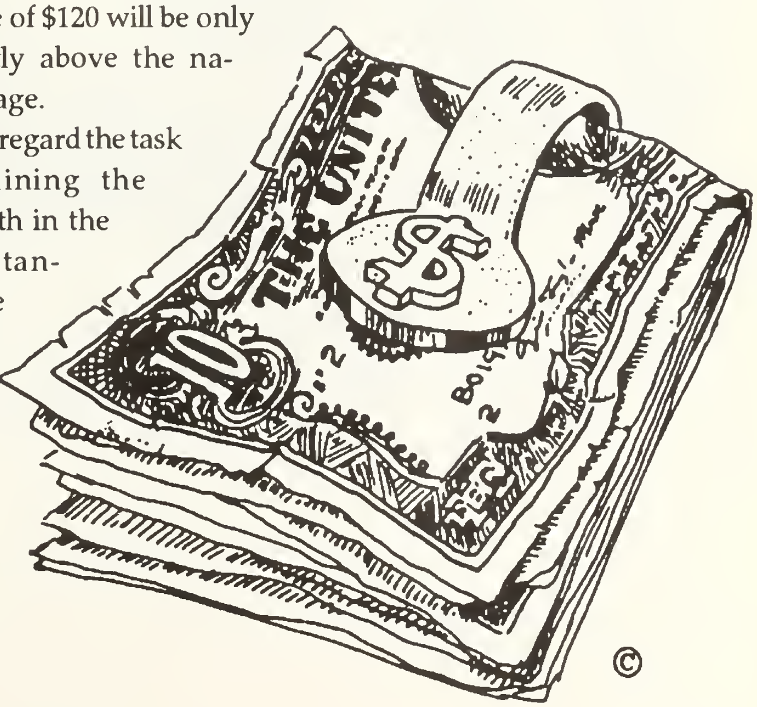
Some physicians have asked why, if our task is to protect the public, not physicians, funding doesn't come from the state's general revenue rather than from physicians. Historically, practice as a physician (or, for that matter in any learned profession) has been treated as a privilege, with the profession bearing the cost and responsibility for public accountability for its professional standards.

In investigating the equality of the renewal fee, we calculated the average fee for all other state boards for last year. It was $106. Our fee was $75 last year. We don't know how much fees will rise in other states but it is clear that our fee of $120 will be only very slightly above the national average.

Fig. 1: Numbers of Complaints in Missouri

1987	381
1988	386
1989	489
1990	741
1991(est)	885

We regard the task of maintaining the public's faith in the quality standards of the profession as being a very important task. We ask for your continued support at a time when the public is questioning the profession's commitment to these professional standards.



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Brief Summary. Consult the package insert for complete prescribing information.

Indications and Usage: 1. *Active duodenal ulcer*– for up to 8 weeks of treatment at a dosage of 300 mg h.s. or 150 mg b.i.d. Most patients heal within 4 weeks.

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3. *Gastroesophageal reflux disease (GERD)*–for up to 12 weeks of treatment of endoscopically diagnosed esophagitis, including erosive and ulcerative esophagitis, and associated heartburn at a dosage of 150 mg b.i.d.

Contraindication: Known hypersensitivity to the drug Because cross sensitivity in this class of compounds has been observed, H_2-receptor antagonists, including Axid, should not be administered to patients with a history of hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*–1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests–False-positive tests for urobilinogen with Multistix® may occur during therapy

Drug Interactions–No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently

Carcinogenesis, Mutagenesis, Impairment of Fertility–A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups.The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy–Teratogenic Effects–Pregnancy Category C–Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers–Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use–Safety and effectiveness in children have not been established.

Use in Elderly Patients–Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic–Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular–In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS–Rare cases of reversible mental confusion have been reported.

Endocrine–Clinical pharmacology studies and controlled clinical trials showed no evidence of anti-androgenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic–Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported

Integumental–Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity–As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other–Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

PV 2093 AMP [101591]

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YOCON®

YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

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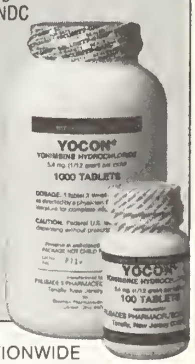

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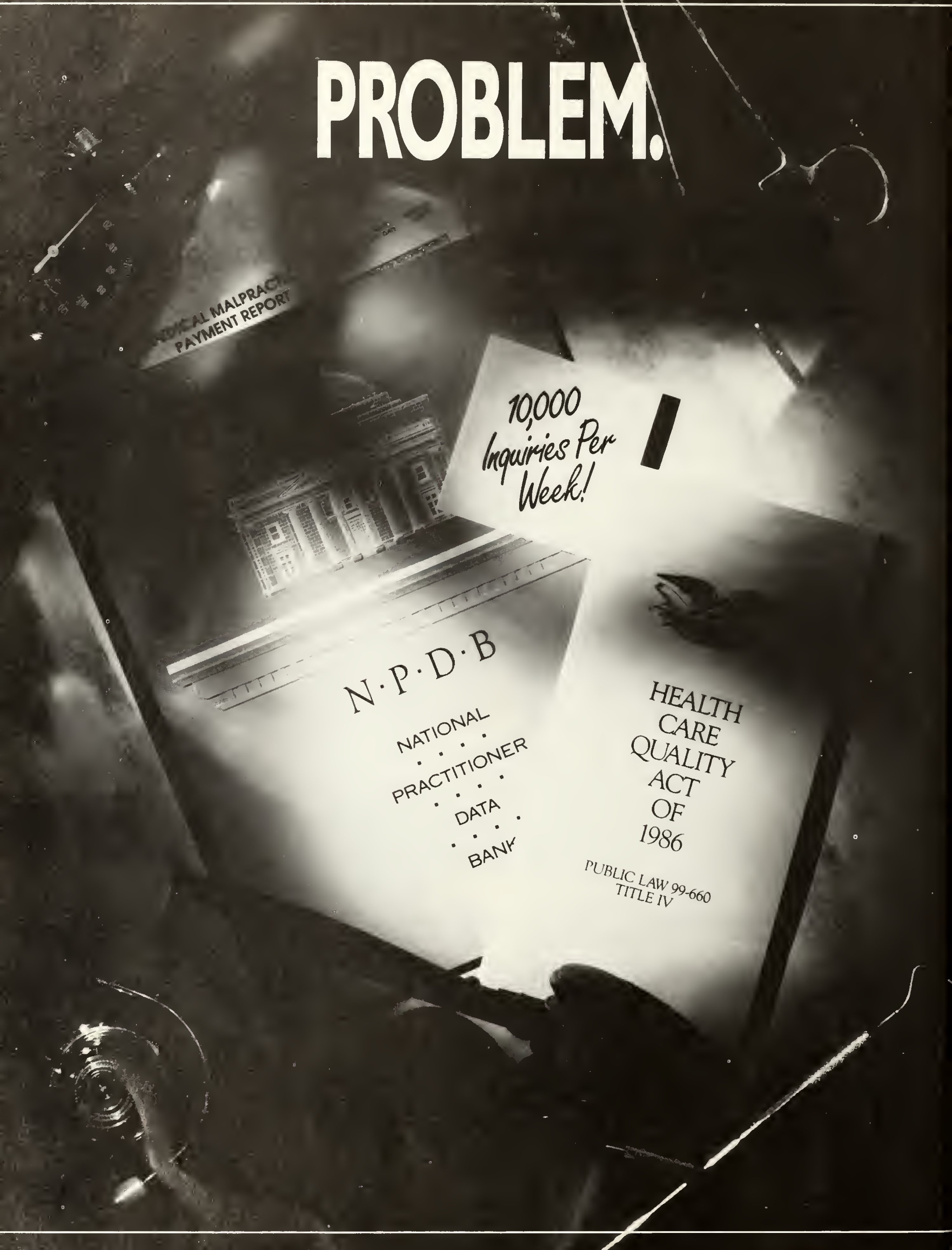
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In Memoriam

■ **Eugene H. Ebel**, M.D., died November 15, 1991 at the age of 68. Dr. Ebel, a retired pediatrics specialist, practiced in north St. Louis county from 1954 to 1986. He had been on the staff at Cardinal Glennon Children's Hospital, Christian Hospitals Northeast and Northwest and was head of pediatrics at DePaul Health Center in Bridgeton. He graduated from the St. Louis University School of Medicine in 1947 and later taught there for more than 20 years. He served in the Navy in World War II and with the Air Force in the Korean War. Dr. Ebel was active in the Daily World Missionaries, a Catholic charity, and was a member of the St. Louis Metropolitan Medical Society.

■ **Alfred Fleishman**, M.D., died November 20, 1991 at the age of 80. Dr. Fleishman was in private practice for 43 years, specializing in internal medicine and gastroenterology. He was on the staffs of Jewish, Barnes and Missouri Baptist hospitals. Co-founder of the Overland Medical Center in Overland and long-time member of the American Medical Association, Dr. Fleishman earned his bachelor's and medical degrees from Washington University. He served in the Army Medical Corps during World War II. Dr. Fleishman was a member of the St. Louis Metropolitan Medical Society.

■ **Joseph A. Bauer**, M.D., died October 28, 1991 at age 89. Dr. Bauer was born in St. Louis and received his medical degree from Washington University School of Medicine. Specializing in pediatrics, Dr. Bauer was on staff at St. Louis Children's Hospital for more than 40 years and was recently honored by the hospital for his service there. Throughout his career, he volunteered in the area's public health clinics, and after retiring in 1963, he served as the physician for the Clayton School District. Dr. Bauer was a member of the St. Louis Metropolitan Medical Society.

■ **Herbert M. Arnold**, M.D., died November 11, 1991 at age 84. Dr. Arnold graduated from what is now SMSU in 1940 and served for a time as a teacher and principal in public schools at Morrisville and Bolivar. In 1974, he was graduated from the University of Kansas Medical School, Kansas City, KS. Following internship at University of Kansas Medical Center, he moved to Lamar, Missouri where he practiced at the Lamar Clinic until his retirement in 1989. Dr. Arnold was a member of the American Society of Contemporary Medicine and Surgery, American Medical Association, American Academy of Family Practice, Missouri State Medical Association, and the Barton County Medical Society.

■ **Ivan A. Anderson**, M.D., died November 8, 1991 at age 88. Dr. Anderson served in private practice in Filer, Idaho, before coming to Columbia in 1965, when he began work as a physician at the University of Missouri-Columbia's Student Health Service. He was also an assistant professor of Community Health and Medical Practice. Dr. Anderson retired from the university in 1973 after a 36 year medical career. He was a member of the Boone County Medical Society.

■ **Calvin J. Curts**, M.D., died October 30, 1991. Dr. Curts retired earlier this year. He was a staff member at St. Luke's Hospital, where he was chairman of the ophthalmology department from 1979 to 1985. He was a clinical associate professor at the University of Missouri-Kansas City and the University of Kansas. He graduated from University of Kansas School of Medicine in 1947 and obtained board certification in ophthalmology in 1956. He was a member of the American College of Surgeons, American Medical Association, Missouri Ophthalmological Society Inc., the Missouri State Medical Association and the Metropolitan Medical Society of Greater Kansas City.

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Bronchopulmonary Lavage for Pulmonary Alveolar Proteinosis

Kara Tate, M.D., Patrick Zoellner, M.D., and Eugene T. Bode, M.D.

Pulmonary alveolar proteinosis is a rare disease.[1] Patients with this disease typically become short of breath and show a diffuse five-lobe infiltrate radiographically. The treatment of choice is total lung bronchopulmonary lavage. Two cases of pulmonary alveolar proteinosis, treated with a total of five bronchopulmonary lavage procedures, are described, along with our lavage apparatus. Following therapy, both patients were able to resume active lives with clinically improved pulmonary status within one week of treatment.

Methods

The patients were brought to the operating room, where a radial arterial line was placed along with other standard monitors, including ECG, pulse oximetry, O_2 analyzer, end-tidal CO_2, temperature probe and a peripheral nerve stimulator. Induction of general anesthesia was accomplished using intravenous fentanyl and thiopental with succinylcholine to facilitate intubation. A left-sided Robert-Shaw double lumen endotracheal tube (ETT) was positioned; position was confirmed bronchoscopically. With the patient in the supine position, the lavage apparatus (Fig 1) was connected to the double lumen tube. The desired lung was lavaged with 500 to 1,000 mL increments of normal saline with heparin (0.1 units per mL). The volume of lavaged fluid was measured and recorded to assure near total return of infused volume. Periodic chest percussion and position changes were performed to aid drainage of the fluid. Anesthesia was maintained with isoflurane, and additional muscle relaxation obtained with vecuronium. Controlled ventilation with a FiO_2 of 1.0 was used throughout. Continuous pulse oximetry and frequent arterial blood gas analysis were used to insure adequate oxygenation and ventilation. The procedure was concluded when the effluent was judged to be non-turbid. The double lumen ETT was then changed to a single lumen ETT. Chest percussion was continued every hour for 2 to 3 hours, and patients were extubated within 2 to 5 hours after the procedure.

Our tubing arrangement is similar to that described by Rogers, et al.[7] Our particular arrangement allows for maintenance of a closed sterile system, which may reduce the risk of introducing infection (Fig 1). The reservoir was suspended approximately 50 cm above the chest to promote alveolar filling in less compliant lung zones.[2,6,11]

The polyvinylchloride tubing from the reservoir is the T-U-R Y-set from Abbott Laboratories, designed for cystoscopy. The tubing has an internal diameter of 0.243 inches and length of 94 inches. During lavage, limbs C and D are clamped. The tubing from the reservoir, limb A, is joined to Y-connector no. 1. From this Y-connector, fluid continues down limbs B and B′ into the lavaged lung via the ETT. Limb B is a cut segment of the T-U-R Y-set tubing, which joins Y-connector no.1 with Y-connector no.2. Limb B′ represents the leg of Y-connector no.2 joining limb B with the ETT. Limb C is the other leg of Y-connector no.2; it permits ventilation of the lavaged lung. Limb B is clamped if ventilation of both lungs is desired. During drainage, limb C remains clamped while limb A is clamped and limb D unclamped. This allows fluid to drain from limbs B′ and B back to Y-connector no.1 and down limb D into a reservoir for measurement and analysis. Limb D is standard urinary reservoir tubing. This system allows for quick transition between draining and filling phases of the lavage. This tubing arrangement is easily assembled and readily available in most hospitals and is mechanically similar to that described by Rogers. The system can be gas sterilized and reused.

Case No.1

A 27-year-old, 83-kg man had a 3-month history of chronic dry cough and increasing dyspnea on exertion. History and physical examination were unremarkable and his chest x-ray film revealed a five-lobe infiltrate. Bronchoscopic washings showed extensive periodic acid Schiff (PAS)-positive material consistent with pulmonary alveolar proteinosis (PAP). An open lung biopsy confirmed the diagnosis. A ventilation-perfusion scan revealed 60% ventilation to the right and 40% to the left lung. Perfusion was uniform throughout. Pulmonary function tests indicated a mild restrictive pattern. In view of increasing symptoms and the distribution of ventilation, the patient was prepared for left total lung lavage.

Under general anesthesia, a 37 french left sided Robert-Shaw double lumen ETT was positioned bronchoscopically. The lavage apparatus was connected to the double lumen tube and the left lung lavaged using normal saline with heparin (0.1 units per mL) in 500 mL to 1,000 mL increments. The volume of infused fluid and the volume drained were carefully recorded to confirm return of the entire volume infused.

Oxygen saturation varied from 98% with the lung full of lavage fluid to 92% with the lung empty. Periodic chest percussion and position changes were performed to aid drainage of the lavaged lung. Total lavage volume was 9,500 mL in and 9,250 mL out. When the effluent was judged to be non-turbid, the double lumen ETT was changed to a single lumen tube. Chest percussion was continued every hour for three hours post-operatively, and the patient was extubated four hours after the procedure.

The right lung was lavaged 48 hours later using a similar technique. The procedure was terminated after infusion of 11,000 mL of lavage fluid. All 11,000 mL were recovered from the lung. Oxygen saturation varied from 95% to 98% with emptying and filling of the lung. Chest percussion was performed periodically during the procedure and every hour for three hours post-operatively. The patient was extubated four hours after the procedure and was discharged from the hospital in good condition 36 hours following the second lung lavage.

Case No.2

A 26-year-old woman with a previous diagnosis of PAP experienced increasing shortness of breath and an increase in appearance of alveolar infiltrates on chest film. She had been treated successfully with bilateral lung lavage approximately three years previously. Her history was significant only for this previous episode. Pulmonary function tests were consistent with a mild restrictive pattern. In view of her increasing symptoms a repeat pulmonary lavage was scheduled.

Under general anesthesia, she was intubated with a size 37 French left sided Robert-Shaw double lumen ETT. Left lung lavage was initiated using normal saline with heparin (0.1 units/mL) in 1,000 mL increments. The effluent was consistently less than the volume infused by 100-200 mL per liter of lavage. After infusion of 12 L of lavage fluid, 1,800 mL of fluid failed to return. The procedure was terminated even though the drainage fluid was still turbid. Oxygen saturation remained in the 98-100% range throughout the procedure.

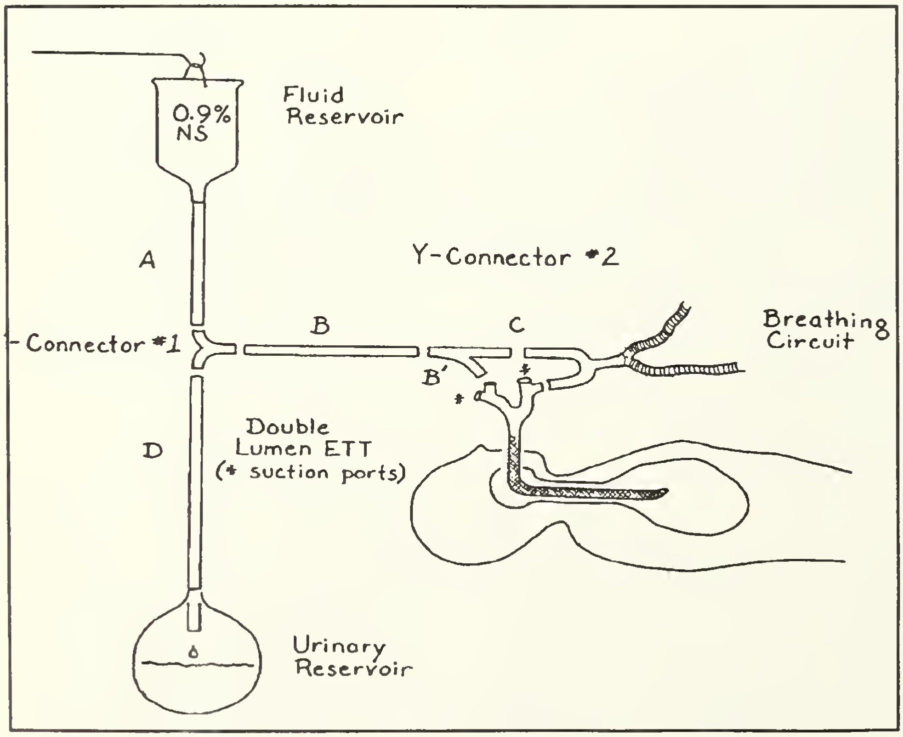
A post-operative chest x-ray film showed dense alveolar consolidation in the left lower lobe consistent with a left pleural effusion. The patient was clinically stable with adequate oxygen saturation and was extubated five hours post-operatively.

Over the next 24 hours the pleural effusion expanded. A thoracentesis was performed and 1,200 mL of clear fluid was drained from the pleural space. No further effusion developed.

The patient was returned to the operating room the following day for lavage of her right lung. Increments of 500 mL to 1,000 mL normal saline with heparin were used for a total volume of 10,500 mL. With intermittent chest percussion to aid drainage, 10,500 mL of fluid was retrieved with progressively increasing clarity. Oxygen saturation fluctuated between 94% with the lung full and 87% with the lung empty. She was extubated three hours after the procedure with adequate gas exchange.

Two days later a third lung lavage was performed in an attempt to complete the aborted lavage of the left lung. This procedure was completed using a total volume of 6,000 mL. The effluent cleared, and a total of 5,900 mL of fluid was retrieved.

The patient was extubated two hours after the procedure and recovered uneventfully. She was discharged from the hospital 24 hours following the third procedure.



Discussion

Pathophysiology

Pulmonary alveolar proteinosis was first described in 1958 by Rosen.[1] Although much has been written regarding the pathology, diagnosis and clinical features of this disease, the cause remains unknown. The pulmonary changes likely represent a final common pathway for a multitude of disease states and environmental insults. Diagnosis is established by open-lung biopsy and substantiated by clinical and roentgenographic features. Microscopically, PAP is characterized by an intra-alveolar, extracellular accumulation of proteinaceous eosinophilic material. The major component of this substance is a phospholipid that stains PAS-positive, a diagnostic feature of the disease. The origin of the intra-alveolar material is felt to be surfactant.[2] Type II pneumocytes synthesize alveolar phospholipids, including lung surfactant, and remove surfactant from the alveolar space. Pulmonary alveolar proteinosis may result from excessive production of phospholipids by the type II pneumocyte, abnormal alveolar macrophage clearance, or a combination.[3]

Clinical Features

The most prominent clinical feature of PAP is dyspnea. Other findings include dry cough, fever, hemoptysis, pleuritic chest pain, clubbing of the fingers, and cyanosis. Laboratory features include a restrictive pattern on pulmonary function tests, decreased lung diffusing capacity (DLco), paO_2, and SaO_2. An elevated LDH has also been associated with active disease.[4] Chest radiograph findings are described as bilateral symmetric alveolar infiltrates, but many variations are recognized.[5] Our patients experienced dyspnea as the primary symptom and demonstrated a restrictive pattern on pulmonary function tests, decreased paO_2, and exercise desaturation with bilateral alveolar infiltrates on radiograph. One patient had a mild elevation of LDH.

Treatment

Currently, total-lung bronchopulmonary lavage (BPL) is the most efficacious mode of therapy for PAP. The use of a double-lumen ETT permits complete separation of the ventilated lung from the lavaged lung. Bronchopulmonary lavage does not alter the underlying disease process, but does mechanically remove intra-alveolar phospholipids. Steroids, potassium iodide, antibiotics and streptokinase, as well as aerosolized saline, heparin, trypsin and acetylcysteine have all been used in an attempt to mobilize the proteinaceous material, with variable success.[6]

Risks

There have been numerous reports of successful bronchopulmonary lavage, though the procedure is not risk-free. Hypoxemia represents the greatest hazard. To reduce the chances of developing hypoxemia, high concentrations of inspired oxygen (FiO_2 1.0) may be used, with pulse oximetry and frequent arterial blood gas (ABG) analysis. The distribution of ventilation and perfusion to each lung should be determined preoperatively, and the more affected lung lavaged first.

We described the paO_2 changes during the filling and emptying of the lavaged lung. The paO_2 rises when the lavaged lung is filled with saline and drops when the lung is emptied. Rogers found filling of the lung was associated with a decreased right to left shunt. During filling, airway pressure exceeds pulmonary artery pressure, diverting blood from the lavaged lung to the ventilated lung.[7] By limiting the time the lavaged lung is empty, the risk of hypoxemia may be decreased.

Other risks of bronchopulmonary lavage include pneumothorax, hydrothorax, hypotension, drainage of fluid into the opposite lung and electrolyte disturbances due to hemodilution resulting from absorption of lavaged fluid. One of our patients developed a hydrothorax requiring thoracentesis.

Rogers found that changes in mean arterial pressure, cardiac output, and arteriovenous O_2 content were due primarily to anesthetic factors rather than the phase of lavage.[7] Impaired venous return may cause hypotension due to mediastinal shift during filling of the lung.[9] The increase in central venous pressure and left ventricular end diastolic pressure that may be noted during lavage is likely a reflection of the transmitted pressure from alveolar filling and not an indication of left ventricular failure.[10] Drainage into the opposite lung can be minimized by careful placement of the double-lumen ETT and elimination of air leaks before beginning lavage. Though theoretically possible, absorption of fluid and electrolyte disturbances are not clinically significant problems.[8]

Other Considerations

The need for central venous monitoring during bronchopulmonary lavage has been questioned. We used a pulmonary artery catheter during our first lavage, but we felt the information it provided did not warrant the risk and cost of its use. All four of our subsequent lavages were performed without central venous monitoring and no untoward sequelae were noted. Other investigators have reached similar conclusions and reserve central monitoring for patients who are at high risk of complications, such as those with decreased left ventricular function or pulmonary hypertension.[2,11] We feel, however, that an arterial line is essential for detection of sudden hemodynamic changes during the procedure, and for ABG analysis.

Various investigators have discussed the positioning of the patient to maximize clinical efficacy of the treatment. Costello recommended avoiding lavage of the dependent lung in the lateral position to prevent gravitational redistribution of blood flow to the dependent lung.[12] Lipmann and Mok advocate the supine position.[10] Harrison and Kelly performed bronchopulmonary lavage in the lateral position with the lavaged lung in both the dependent and nondependent positions. With lavage of the dependent lung, there was less risk of drainage into the ventilated lung, while ventilation of the nondependent lung was less compromised by mediastinal movement and the weight of the saline-filled lung. Lavage of the nondependent lung offers the advantage of diverting pulmonary blood flow to the dependent, ventilated lung as well as making drainage easier.[9]

We elected to use the supine position in conjunction with rotation of the table and chest physiotherapy to aid in removing debris and fluid. No significant problems with hypoxemia or leakage of fluid were noted in our patients.

The fluid to be lavaged must be isotonic (usually normal saline) and warmed to 37°C to prevent hypothermia. The addition of heparin, acetylcysteine or trypsin to the saline may reduce the viscosity of secretions. We elected to use normal saline with heparin, 0.1 μ/mL. Plain normal saline has been reported to be as effective as solutions containing heparin or acetylcysteine.[13]

Summary

In summary, total-lung bronchopulmonary lavage was performed five times on two patients under general anesthesia with controlled ventilation using a Robert-Shaw double lumen ETT. One lavage was complicated by a hydrothorax that was treated without untoward sequelae. No significant hypoxemia, circulatory impairment or leakage of lavaged fluid into the opposite lung was noted on any occasion. The average time of all procedures was 108 minutes (range 80-150 minutes), and patients were extubated on all occasions 2 to 5 hours after the treatment. Patients were discharged 24 to 36 hours after the last lavage with improvement, though not resolution, of the alveolar infiltrates radiographically. The ABG analysis revealed minimal improvement in oxygenation in Case No.1 from a preoperative paO_2 of 60 mmHg on room air to a paO_2 of 74 mmHg on the day of discharge. However, the patient was able to resume his normal activity level. In Case No.2, the patient's resting paO_2 did not improve significantly from the preoperative value of 82 mmHg. Subjectively, however, the patient noted marked improvement. Prior to bronchopulmonary lavage, this patient's SaO_2 decreased from 97% to 81% after walking 800 feet. Approximately a week and a half after discharge, the patient reported being able to run without symptoms and generally was feeling well.

With careful attention to detail and the coordination of resources from the departments of anesthesia, pulmonary medicine, respiratory therapy and nursing services, bronchopulmonary lavage can be performed safely and efficiently in order to curtail the progressive hypoxemia which often develops in patients with PAP.

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Massive Gastric Hemorrhage due to Small Vessel Erosion (The Dieulafoy Lesion)

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Columbia, Missouri

With the widespread use of endoscopy, the Dieulafoy gastric lesion, a cause of major upper gastrointestinal bleeding, is being recognized with increasing frequency. The lesion was first described in 1884 by Gallard[1] and was more completely characterized by Dieulafoy in 1897.[2] The lesion, usually a small "grain of rice"-sized area of mucosal erosion that penetrates an artery, has been the subject of a number of reports.[3-9] The artery affected is usually larger and more superficial than surrounding vessels. The clinical picture includes massive blood loss, melena, and, often, hematemesis. Typically, there is no history of peptic ulcer disease or gastritis. The cause of the erosion is unknown.

We report a case in which the patient had had repeated upper GI endoscopy before an episode of massive gastric bleeding. Initially, the bleeding was mistakenly attributed to aspirin-induced gastritis.

Case Report

The patient was a 62-year-old physician who was admitted with a 12-hour history of orthostatic faintness and dyspnea on exertion. The patient had noted dyspnea with minimal exercise the evening before admission. The following morning he felt lightheaded upon arising and dyspneic during routine activity. There had been a slight darkening of the stool during the preceding few days but no frank melena. His medical history was significant in that he had had gastroesophageal reflux with resultant scarring and stricture formation at the distal end of the esophagus. He experienced periodic difficulty with swallowing, which had been alleviated by dilatation of the stenotic esophageal gastric junction. During the two-year period before admission, the patient had undergone upper GI endoscopy six times, most recently four months before admission. The patient followed an antireflux regimen that included elevation of the head of the bed, liberal use of antacids, and 150 mg of ranitidine twice daily. The discomfort of degenerative joint disease prompted the patient to use buffered aspirin, two 325 mg tablets, one to three times daily. For a number of years the patient had had mild hypertension controlled with a variety of medications including propranolol, hydrochlorothiazide, and enalapril.

On entry to the emergency aoom the patient was pale, slightly dyspneic, and complained of lightheadedness when standing. His vital signs included supine pulse, 110 beats/minute; standing pulse, 140 beats/minute; blood pressure, 140/80 supine, 80/60 standing; respirations, 20/minute; temperature 98.6°F. Results of the physical examination were essentially normal. Stool obtained on rectal examination was dark brown and tested strongly positive for blood. Initial laboratory values obtained in the emergency room included a hematocrit of 33%, Hgb of 12.9 g/dL, and a BUN of 36 mg/dL. The patient was admitted with a diagnosis of gastrointestinal bleeding, site unknown. An intravenous infusion of saline solution was started, and his blood typed and crossmatched in preparation for possible transfusion.

Initial attempts to pass a nasogastric tube were unsuccessful, but did induce vomiting of bright red blood, indicating the likelihood of an upper GI bleeding site. Within six hours of admission, the patient underwent esophagogastroduodenoscopy (EGD). The esophagus appeared free of stricture and the duodenum was normal. The lumen of the stomach was obscured by clots of blood. The endoscope was withdrawn, a gastric tube passed, the stomach lavaged with 500 mL normal saline, and the endoscope reintroduced. A pool of bright red blood was still present, but diffuse hemorrhagic gastritis of the body and cardia of the stomach was noted in the areas free of blood. The gastritis was felt to be secondary to aspirin use. The patient was treated with antacids, a slurry of sucralfate, and intravenous ranitidine. During the first hospital day, the patient's hemoglobin fell to 8.8 gm/dL and the hematocrit to 25%. The BUN decreased to 24 mg/dL; the patient's chemistry profile was otherwise normal.

The patient was given two units of packed red blood cells the evening of admission. After this, the hemoglobin and hematocrit rose to 9.7 gm/dL and 28% respectively. The patient remained comfortable without nausea or emesis. The stool became melanotic for two days, then returned to a normal brown color.

By the fourth hospital day the patient was no longer faint on standing. His hemoglobin dropped to 8.1 gm/dL. This change was felt to be due to rehydration rather than to active bleeding. He was discharged home on a regimen of antacids, ranitidine, and sucralfate. The patient was to return in 48 hours, or sooner if there was evidence of renewed bleeding.

During the evening after discharge, symptoms of orthostatic faintness and mild dyspnea returned and over the next 12 hours, worsened. The patient had one dark brown, sticky stool. He reported to the emergency room, where he was found to have a blood pressure of 160/80 supine, and 90/60 standing. His pulse was 130 beats/min standing; his hemoglobin was 7.6 gm/dL and the hematocrit 22%. Over the ensuing four days he received 11 units of packed red blood cells. He continued to take antacids, sucralfate, and ranitidine. On the morning of the third hospital day, upper GI endoscopy was repeated. The diffuse hemorrhagic gastritis noted six days earlier had disappeared. The gastric mucosa appeared normal except for a 2 mm area on the posterior wall of the greater curvature in the cardia that was oozing blood. Despite transfusions of packed red blood cells, the patient's hematocrit failed to stabilize. He passed liquid black stools and continued to experience faintness on standing. In the late afternoon of the third hospital day, a nasogastric tube was passed and bright red blood aspirated. The stomach was thoroughly lavaged with ice water, the gastric tube was left in place, and the patient was transferred to the medical intensive care unit. Through the night a slurry of sucralfate was instilled hourly via the gastric tube. Despite these efforts, active bleeding persisted as indicated by melanotic stools, orthostatic changes in the blood pressure and pulse, and a low hematocrit that did not improve with the repeated transfusions. In the late afternoon of the fourth hospital day, the patient underwent endoscopy for the third time. The small erosion on the posterior wall of the greater curvature in the cardia was seen. In its center was a small artery spurting blood. Efforts to stop the bleeding by electro-coagulation were unsuccessful. The decision was made to carry out an emergency gastrostomy in order to oversew the eroded vessel. The location of the erosion was noted, the endoscope withdrawn, and the patient prepared for surgery. Early on the fifth hospital day the patient was taken to the operating room, the stomach was opened and the eroded vessel located and ligated. Except for a small clot over the suspected artery, the stomach was free of blood and the gastric mucosa appeared normal. There was no evidence of gastritis. Bilateral vagotomy and pyloroplasty were carried out without incident.

The post-operative course was uncomplicated. There was no further bleeding. The patient was discharged on the sixth post-operative day with an hematocrit of 32%. During the four years since discharge, the patient has taken ranitidine 150 mg twice a day, has not required antacids, and has avoided non-steroidal, anti-inflammatory drugs, including aspirin.

His difficulties in swallowing have recurred periodically, prompting endoscopy and dilatation on eight occasions, the most recent being September 1991, nearly four years after the bleeding episode. Inspection of the stomach on each occasion has revealed completely normal gastric mucosa.

Discussion

After the common causes of massive upper GI bleeding have been excluded, there remain some 5% to 10% of cases that are candidates for the diagnosis of Dieulafoy's disease. Typically, these cases have a number of features in common, that is, the absence of a significant medical history of peptic ulcer disease, analgesic abuse, or alcoholism. The bleeding ensues rather abruptly, is unremittent with conservative treatment, is associated with a high mortality rate, and rarely recurs. Pathologically, there is a small area of erosion that penetrates the submucosa and muscularis layers and is associated with the rupture of a small gastric artery. The location of the erosion is usually within 10 cm of the esophageal-gastric junction, in contrast to the distal bleeding associated with peptic ulcer. In our case, during the third endoscopy procedure, a ruptured artery high on the posterior wall of the greater curvature was seen spurting blood.

The cause and pathogenesis of the Dieulafoy lesion remains unclear. Because of this, a variety of labels have been applied to what is probably the same pathological process. Dieulafoy himself used the term "ex uleratio simplex." Subsequent authors have considered the lesion to be a vascular malformation, an aneurysm, an atherosclerotic process, or an unusually placed peptic ulcer. Miko and Thomazy[10], after careful anatomic analysis, termed the lesion "caliber persistent artery of the stomach." Until more specifics are known, it is reasonable to continue to use the term "Dieulafoy lesion."

Although upper GI endoscopy is a major asset in the diagnosis and management of upper GI bleeding, it is not without pitfalls, as our case report shows. Perhaps the pool of bright red blood in the stomach following the initial lavage was a clue that something more than bleeding from gastritis was occurring. Another clue, not present in our case, but associated with such lesions, is a stalactite or pencil clot emanating from the bleeding site.

Because of the patient's history of aspirin use and the presence of diffuse gastritis during the first endoscopy, the bleeding was erroneously attributed to hemorrhagic gastritis. With the widespread use of analgesics, it isn't surprising that gastritis secondary to analgesic use could co-exist with other less common, more threatening causes of upper GI bleeding. Similarly, peptic ulcer disease may occur coincidentally with the Dieulafoy lesion.

That treatment of Dieulafoy upper GI bleeding has been less than satisfactory is reflected by the high mortality figures. Goldman[11] reported a mortality figure of 30% in 1964. In a series of 35 cases collected after 1970, Veldhuyzen Van Zanten et al[3] reported a death rate of 23%. Deaths occur post-operatively because of failure to locate the bleeding site. Thus, a critical role of endoscopy would be to pinpoint the bleeding site before surgery.

In some centers, treatment with endoscopy alone has had a high success rate. For example, Pointer et al,[12] using local injection of a sclerosing agent or electrocoagulation terminated bleeding in 18 of 22 patients. This high success rate is tempered by the vagaries of endoscopic definition of this lesion. ■

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Reprints

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Racial Differences in Cardiovascular Disease Mortality and Risk Factors

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Cardiovascular disease (CVD), which is comprised mainly of coronary heart disease and cerebrovascular disease (stroke), is the major cause of death, illness, disability, and medical costs in the United States and in Missouri.[1,2] Nationally, CVD claims nearly a million lives per year, or approximately 45% of all deaths.[2] The yearly cost of CVD in the United States is estimated at $101 billion.[2] In Missouri, a total of 22,304 CVD deaths were recorded in 1989.[3] This represented 44% of all deaths for that year.

Over the past 40 years, mortality rates for CVD have declined dramatically. Coronary heart disease rates have decreased 46% and rates of stroke have declined 66%.[4] The causes of the decline in CVD death rates are related both to improvements in medical care and to decreases in CVD risk factors.[5] But despite these overall successes, not all segments of the population have benefitted equally. For example, among white males, death rates for heart disease declined 42% between 1950 and 1988.[4] In comparison, the decrease among black males for the same time period was only 31%.[4] On a national level, it has been observed that more than 7,000 fewer blacks would die of CVD each year if their death rate was the same as that for whites.[6] It has also been noted that epidemiologic data on the impact of CVD on minority populations are relatively sparse.[7]

A large body of clinical and epidemiologic evidence has identified numerous risk factors for CVD. The three major CVD risk factors are cigarette smoking, high blood pressure, and high blood cholesterol.[1,8] Several other controllable risk factors have been implicated in CVD etiology. These include physical inactivity, obesity, diabetes, and stress.[1,8,9] In addition to these modifiable risk factors, non-modifiable CVD risk factors include age, male gender, black race, and family history of CVD.[1,9]

A variety of population-based strategies have successfully targeted the major CVD risk factors of smoking, high blood pressure, and high blood cholesterol.[6] Growing attention has also been focused on other CVD risk factors such as physical inactivity and obesity.[8] Although risk factor control strategies have been successfully developed for many sociodemographic groups, high risk populations (i.e., minorities and the economically disadvantaged) have not always received the level of attention warranted by the magnitude of the CVD problem.[10]

An earlier *Missouri Medicine* article[11] discussed the overall impact of CVD in Missouri. In this paper, we compare CVD mortality rates and common CVD risk factors between blacks and whites in Missouri. Examination of the racial distributions of various CVD risk factors provides the scientific basis for developing and targeting control strategies for high-risk populations.

Methods

Mortality Analyses

Data from the Missouri Center for Health Statistics[12] were used to examine the magnitude of cardiovascular disease in 1989. The category of all cardiovascular diseases was encompassed in *International Classification of Diseases, 9th Revision (ICD-9)*[13] codes 390-448.

Behavioral Risk Factor Data

Since 1985, the Missouri Department of Health has been collecting data on the prevalence of behavioral risk factors such as smoking, high blood pressure, high blood cholesterol, physical inactivity, and obesity.[14,15] This surveillance system, sponsored by the Centers for Disease Control, uses a random digit dialing technique[16] to select adult Missourians, age 18 years and older, for telephone interviews. The interviews are conducted monthly, and a total of 6,146 interviews (from January 1987 through April 1991) were available for this analyses. Not all questions on each risk factor were asked in all years; therefore, the total sample size for each CVD risk factor varies slightly. In order to examine racial differences in risk factors, as many years as possible were pooled. Small sample sizes among racial groups other than whites precluded examination of time trends in CVD risk factors for these groups. When interpreting our findings, it should be noted that these are self-reported data. The actual population prevalence of these CVD risk factors is likely to be under-reported.

Population Attributable Risk Calculations

In order to assess the overall public health importance of various CVD risk factors, estimates of population-attributable risk percent (PAR%) were calculated.[17] The PAR% is defined as the proportion of a disease that could be prevented if a given risk factor were eliminated.[18] To calculate PAR% estimates, we used published relative risk estimates for various factors[19] and risk factor prevalence estimates based on our statewide behavioral risk factor survey.

Results

The age-adjusted mortality rate per 100,000 for coronary heart disease was 167 among blacks, compared with 119 among whites (Fig 1). This is a black-white difference in mortality of 40%. For stroke mortality, the rates per 100,000 of 46 among blacks and 26 among whites represents a difference of 77%.

Figures 2-6 illustrate behaviors and risk factors that largely account for the elevated rates of CVD mortality among blacks. The prevalence of cigarette smoking was higher among blacks than among whites (Fig 2). The highest smoking rate of 31% was noted for black men, and the lowest rate (25%) was observed among white women.

Figure 3 shows that more women than men, and more blacks than whites reported having high blood pressure. Although no data were available on actual blood cholesterol levels, we can report the percentage of individuals who had never had their cholesterol level checked (Fig 4). These data show that nearly half of the Missouri population had never had their cholesterol level checked. The subgroup least likely to have cholesterol levels checked was black men (50% reported never having their cholesterol checked).

The lack of physical activity is another important CVD risk factor.[19] Figure 5 shows that the majority of adult Missourians are physically inactive (i.e., no recreational physical activity in the month preceding the survey). The prevalence of physical inactivity was higher among women, especially black women (71% were physically inactive). Data on the frequency of overweight (more than 120% of ideal body weight) also showed fairly large black-white differentials (Fig 6).

Discussion

Missouri data on CVD mortality and risk factors show the same general trends as those for the U.S. population.[20,21] For example, in Missouri and in the United States the highest smoking rate was observed for black males (*i.e.*, 31% in Missouri and 33% in the United States).[21]

Although our data show alarming black-white discrepancies in CVD mortality, they also indicate numerous areas (*i.e.*, behavioral risk factors) in which tremendous potential exists for CVD prevention and control.

Estimates of PAR% are perhaps the best indication of preventive capability. The PAR% for coronary heart disease due to cigarette smoking among black males was 22%. This implies that, among black men, mortality due to heart disease could be reduced by 22% through elimination of smoking. The corresponding figure for black females was 19%. In addition, for cigarette smoking among Missouri blacks, data on smoking intensity from our state risk factor survey suggest great potential for smoking reduction. Although the overall rates of smoking were higher in blacks than in whites, blacks were consistently found to smoke fewer cigarettes per day than whites. Among blacks, the percentages who smoked 1-10 cigarettes per day, 11-20 cigarettes per day, and ≤ 21 cigarettes per day were 55%, 37% and 8%, respectively. The corresponding figures for whites were 22%, 48%, and 30%. Numerous studies have shown that cessation efforts are much more effective for light smokers than for heavy smokers.[22]

Estimates of PAR% for other CVD risk factors also indicate a large preventive potential. For physical inactivity and heart disease, the PAR% values for black males and black females were 36% and 39%, respectively.

Although relatively little attention has been focused on developing effective CVD risk reduction programs for minorities,[7] a recent review[6] identified several successful

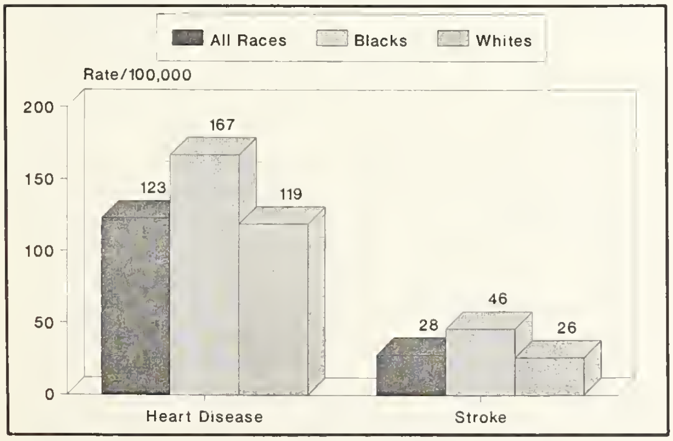

Fig. 1. Age-adjusted rates per 100,000 for coronary heart disease and cerebrovascular disease by race, Missouri, 1989.[12]

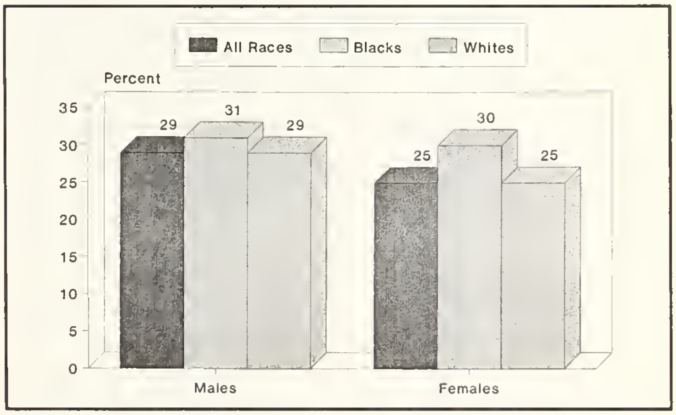

Fig. 2. Estimated prevalence of current cigarette smoking by race and gender, Missouri, 1987-91.[15]

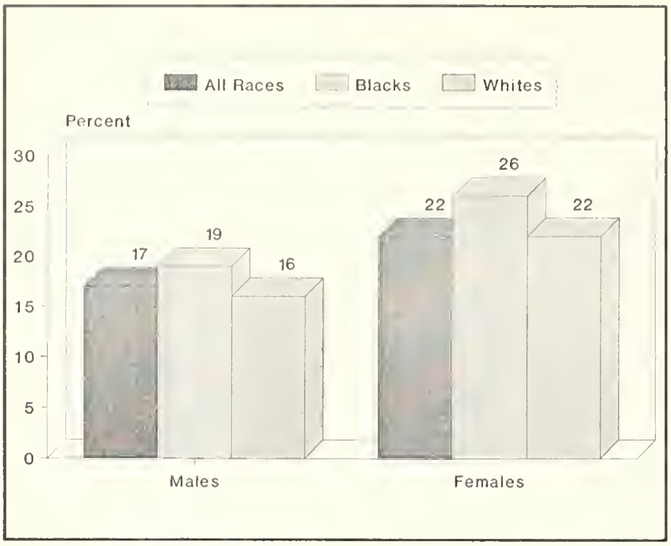

Fig. 3. Estimated prevalence of high blood pressure by race and gender, Missouri, 1987-91.[15]

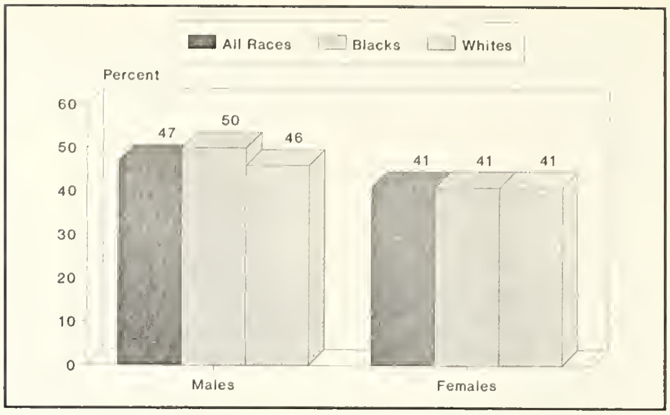

Fig. 4. Estimated prevalence of never having blood cholesterol checked by race and gender, Missouri, 1987-91.[15]

strategies. Frequently occurring themes included:

·Involvement of community leaders in the planning and implementation of programs

·Adaptation of program features to local norms

·Utilization of existing community structures such as churches, barbershops, and social clubs

·Reliance on local voluntary health organizations such as the American Heart Association

We are currently developing community-based coalitions to reduce CVD risk factors in a six-county area in southeast Missouri.[23] Many of these coalitions have adopted the community-based strategies noted above.

The large discrepancies between rates of CVD and associated risk factors in black and white Missourians suggest that a higher priority must be placed on prevention and control of CVD among minorities. This priority is reflected in recent year 2000 health objectives for the nation.[8] Recent action at the state level that will enhance CVD prevention efforts among blacks and other minorities includes the development of community-based coalitions in several areas of the state, and the formation and enhancement of the state Office of Minority Health.

As we noted previously,[11] physicians and other health care professionals are a respected source of information on preventive medicine and can play a key role in achieving year 2000 health objectives. An estimated 80% of the population visits a physician during a given year, and 54% of all encounters are with primary care physicians.[24] These patient-physician encounters are ideal opportunities for prevention of CVD, both primary (e.g., smoking cessation, counseling for physical activity) and secondary (e.g., screening for hypertension and cholesterol levels). However, the situation is more complex for the economically disadvantaged and minority populations that may have limited or non-existent access to primary health care, much less preventive health care.[1]

A systematic public health approach must consider factors beyond mortality rates and CVD risk factor when designing interventions. It is evident that a complex array of issues such as labor, housing, transportation, and social inequities all contribute to an individual's desire and ability to choose a healthier lifestyle.[10] Despite these many considerations and the difficulty of achieving long-term behavior change, there is a large preventive potential for eliminating excess CVD among minorities and other high-risk populations.

Acknowledgements

Joan Wilkerson, Missouri Cancer Registry, Missouri Department of Health, and Bruce Gibson, Missouri Center for Health Statistics, Missouri Department of Health, assisted in data analyses. This study was funded in part by Centers for Disease Control contract U58/CCU700950.

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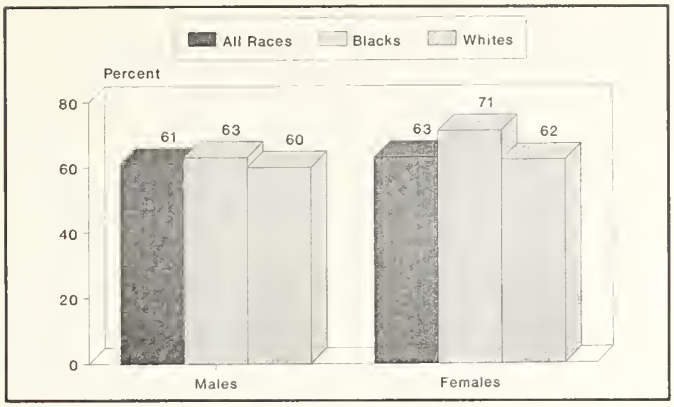

Fig. 5. Estimated prevalence of physical inactivity by race and gender, Missouri, 1987-91.[15]

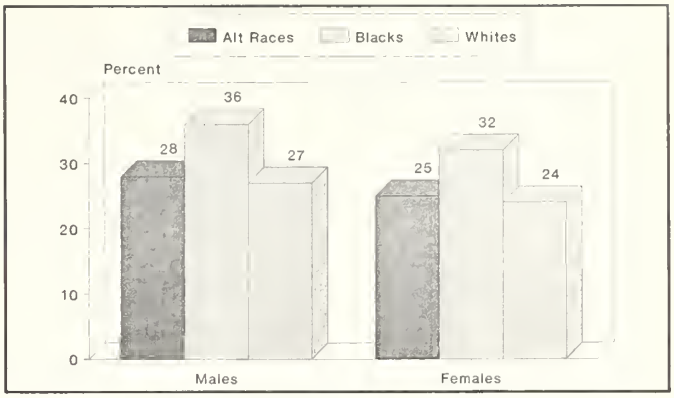

Fig. 6. Estimated prevalence of overweight by race and gender, Missouri, 1987-91.[15]

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Revival of a House Staff Organization

Caroline Kerber, M.D., Karen Zanol, M.D. and the Executive Committee of the University of Missouri-Columbia House staff Organization

The house staff of the University of Missouri-Columbia are in the midst of a revival. Many years ago, the house staff at UMC established and formed a formal organization of unity. It flourished in the late 1970's and early 1980's, but then floundered and dissolved because of lack of leadership and interest. In 1988, a small group of house staff felt they needed better representation and cohesiveness as a group. Formal bylaws were written and edited with final completion and formation of the House staff Organization.

HSO membership is automatic by virtue of being an intern, resident or fellow at University of Missouri-Columbia. HSO serves as the foundation for resident representation in hospital affairs. House staff form a majority of physician manpower at teaching hospitals. HSO Executive Committee is composed of proportional representation from every department. There is resident representation as active members of every major hospital committee from Infection Control to Hospital Executive Committee. Many hospital issues directly or indirectly affect House staff, and proper House staff representation has really prompted better communication with hospital and medical school administration.

HSO supports and promotes involvement in organized medicine at local, state and national levels. Involvement in local HSO sparks interest in young physicians to seek out opportunities for leadership in organized medicine. At UMC, the HSO is currently working hard at resident recruitment to stay competitive in the changing environment of resident education. Additionally, HSO is an active forum for interdepartmental relations and communication, thus promoting a unified house staff.

Future goals include working toward developing community service activities and improving medical student education and recognition of superior faculty. There has never been a more important time for house staff to organize, and the HSO at UMC has never been more positive about the future of graduate medical education.

Note: Please send your program's House Staff Organization minutes or house staff happenings to:

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University of Missouri Hospital and Clinics
Columbia, Missouri 65212

MSMA Patient Brochure Information

MSMA has available several patient information brochures. Topics range from breast cancer treatment alternatives to health care cost containment. You may preview sample copies free of charge before ordering. Submit orders and sample requests, by title, on your professional letterhead, to MSMA Brochures, P.O. Box 1028, Jefferson City, MO 65102. Or, you may fax your orders to the MSMA office at 314/636-8552. Here are just a few of the titles:

A WOMAN'S CHOICE. Presents various breast cancer treatment alternatives in clear layman's language, discussing advantages and disadvantages of each treatment choice. ***Cost: 15 cents each.***

HEALTH CARE COST CONTAINMENT. Do your patients understand why health care costs are rising? Do they realize they can do something about it? In simple language, this popular brochure explains some of the reasons for spiraling costs and shows how the patient and physician working together can help contain them. ***Cost: 10 cents each.***

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UNDERSTANDING MEDICARE. This is a new brochure that explains some of the complexities of the Medicare maze: qualifying, submitting claims, understanding terminology. The information is general and simple, so your Medicare patients and family can benefit. ***Cost: 10 cents each.***

PHYSICIAN'S RECOGNITION AWARDS

Seventy-nine MSMA members qualified for the MSMA-AMA Physician's Recognition Award during October and November 1991. The award, presented by the Missouri State Medical Association and the American Medical Association, is given to those physicians who voluntarily complete 150 credit hours of continuing medical education within a three-year period. Sixty of the credit hours must be earned in Category 1 programs.

Boone

Murray Boles, *Columbia*
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Harold L. Sanders, *Columbia*

Buchanan

H. Brian Balfour, *St. Joseph*
Helen T. Nguyen, *St. Joseph*
Prakashchandra B. Patel, *St. Joseph*
Robert E. Remis, *St. Joseph*

Clay - Platte

John C. Hagan, *Kansas City*

Cole

James L. Rea, *Jefferson City*

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Stephen D. Christiansen, *Springfield*
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Scott W. McMurray, *Springfield*
Robert F. Mahnken, *Springfield*
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SEMO

Laghaieh Rezvani, *Sikeston*

South Central

Laureano G. Mayuga, *Cabool*

St. Louis Metro

Navin J. Amin, *St. Louis*
H. Bradley Binnington, *St. Louis*
Francis J. Catanzaro, *St. Louis*
George M. Bohigian, *St. Louis*
Sophia M. Chung, *St. Louis*
Jonathan D. Dehner, *St. Louis*
Thomas F. Fawley, *St. Louis*
James W. Fleshman, *St. Louis*
Joseph Gagliardo, *Clayton*
Jerome J. Gilden, *St. Louis*
Samuel B. Guze, *St. Louis*
John B. Holds, *St. Louis*
Donald K. King, *Webster Groves*
John E. Krettek, *Chesterfield*
Richard S. Levine, *Creve Coeur*
Dominador O. Madrid, *St. Louis*
John F. Mantovani, *St. Louis*
David B. Marrs, *St. Charles*
Michael E. McCadden, *St. Louis*
Ravi Mehra, *St. Louis*
David M. Near, *St. Louis*
Edward J. O'Brien, *St. Louis*
Margaret M. F. Oakley, *St. Louis*
Hilton I. Price, *St. Louis*
Ronald N. Riner, *St. Louis*
Jitendra P. Shah, *St. Louis*
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New Members

Joseph R. Barnthouse, M.D., 6675 Holmes, Suite 430, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Barnthouse received his baccalaureate degree from the University of Kansas, Lawrence, KS, and obtained his medical degree from the University of Kansas School of Medicine, Kansas City, KS in 1985. He completed his residency at the University of Missouri Truman Medical Center, Kansas City, MO. Dr. Barnthouse specializes in plastic surgery.

Michael F. Bartell, M.D., 1035 Bellevue Ave., Suite 203, St. Louis, MO 63117, has become a member of the Missouri State Medical Association. Dr. Bartell obtained his medical degree from Creighton University School of Medicine, Omaha, NB in 1973. He completed his residency at St. Mary's Health Center, St. Louis, MO. Dr. Bartell specializes in nephrology.

Jonathon M. Bird, M.D., 301 W. Liberty St., Farmington, MO 63640, has become a member of the Mineral Area Medical Society. Dr. Bird received his baccalaureate and medical degrees from the University of Missouri School of Medicine, Kansas City in 1983. He completed his residency at Baptist Medical Center, Kansas City, MO. Dr. Bird specializes in family practice.

Elisa H. Birnbaum, M.D., 216 S. Kingshighway, St. Louis, MO 63110, has become a member of the St. Louis Metropolitan Medical Society. Dr. Birnbaum received her baccalaureate degree from the University of Wisconsin, Madison and obtained her medical degree from the University of Illinois Medical Center, Peoria in 1985. She completed her residency at Long Island Jewish Hospital, Long Island, NY. Dr. Birnbaum specializes in surgery.

Kathryn Botney, M.D., 226 S. Woods Mill Rd., Ste. 68W, Chesterfield, MO 63017, has become a member of the St. Louis Metropolitan Medical Society. Dr. Botney obtained her medical degree from Washington University School of Medicine, St. Louis in 1984. She completed her residency at Washington University Affiliate Hospital, St. Louis. Dr. Botney specializes in obstetrics/gynecology.

Alan C. Braverman, M.D., 4104 Queeny Tower, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Braverman received his baccalaureate degree from the University of Missouri, Kansas City and obtained his medical degree from the University of Missouri School of Medicine, Kansas City in 1985. He completed his residency at Brigham & Women's Hospital, Boston, MA. Dr. Braverman specializes in internal medicine/cardiovascular diseases.

Alan D. Camp, M.D., 12255 DePaul Dr., Suite 865, Bridgeton, MO 63044, has become a member of the Missouri State Medical Association. Dr. Camp received his baccalaureate degree from Grinnell college, Grinnell, IA and obtained his medical degree from the University of Iowa College of Medicine, Iowa City in 1983. He completed his residency at St. Louis University Medical Center, St. Louis, MO. Dr. Camp specializes in internal medicine/cardiology.

Timothy B. Cavanaugh, M.D., 4321 Washington, Suite 600, Kansas City, MO 64111, has become a member of the Missouri State Medical Association. Dr. Cavanaugh received his baccalaureate degree from Southern Methodist University, Dallas, TX and obtained his medical degree from the University of Kansas School of Medicine, Kansas City in 1986. He completed his residency at University Hospital, Kansas City, KS. Dr. Cavanaugh specializes in ophthalmology.

Luis E. Colon, M.D., 621 So. New Ballas Rd., Suite 228, St. Louis, MO 63141, has become a member of the Missouri State Medical Association. Dr. Colon received his baccalaureate degree from the University of Puerto Rico and obtained his medical degree from the University of Puerto Rico School of Medicine, San Juan in 1980. He completed his residency at Fitzsimons Army Medical Center, Aurora, CO. Dr. Colon specializes in ophthalmology.

Joseph A. Crapotta, M.D., 3655 Vista Ave., St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Crapotta received his baccalaureate degree from Columbia University, New York, NY and obtained his medical degree from New York Medical College, New York in 1985. He completed his residency at the Catholic Medical Center of Brooklyn/Queens, Jamaica, NY. Dr. Crapotta specializes in ophthalmology.

Fred T. Creech, III, M.D., 2718 Forum Blvd., Suite 3B, Columbia, MO 65202, has become a member of the Boone County Medical Society. Dr. Creech received his baccalaureate degree from Wabash College, Crawfordsville, IN and obtained his medical degree from Indiana University School of Medicine, Indianapolis in 1980. He completed his residency at the University of Missouri-Columbia. Dr. Creech specializes in dermatology.

John P. Crotty, M.D., 2344 Hampton Ave., St. Louis, MO 63139, has become a member of the St. Louis Metropolitan Medical Society. Dr. Crotty received his baccalaureate degree from St. Louis University, St. Louis, MO and obtained his medical degree from St. Louis University School of Medicine, St. Louis, MO in 1962. He completed his residency at St. Louis City Hospital, St. Louis, MO. Dr. Crotty specializes in radiology.

Christopher T. Cruz, M.D., Anderson Hospital, Rt. 162, Maryville, IL, has become a member of the Missouri State Medical Association. Dr. Cruz received his baccalaureate degree from the University of California, Berkeley and obtained his medical degree from Institute of Medicine Far Eastern University, Manila, Philippines in 1983. He completed his residency at St. Louis University Hospital, St. Louis, MO. Dr. Cruz specializes in internal medicine.

Lucian V. DelPriore, M.D., Box 8096, 660 South Euclid, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. DelPriore received his baccalaureate degree from Cooper Union, New York City, NY and obtained his medical degree from the University of Rochester School of Medicine and Dentistry, Rochester, NY in 1982. He completed his residency at Johns Hopkins University School of Medicine, Baltimore, MD. Dr. DelPriore specializes in ophthalmology.

Todd L. Demmy, M.D., MA 312, #1 Hospital Drive, Columbia, MO 65212, has become a member of the Boone County Medical Society. Dr. Demmy received his baccalaureate degree from Pennsylvania State University and obtained his medical degree from Jefferson Medical College of Thomas Jefferson University, Philadelphia, PA in 1983. He completed his residency at Allegheny General Hospital. Dr. Demmy specializes in cardiothoracic surgery.

Mary K. Downs, M.D., 1000 East Primrose, Suite 310, Springfield, MO 65807, has become a member of the Greene County Medical Society. Dr. Downs received her baccalaureate degree from Rice University, Houston, TX and obtained her medical degree from the University of Texas Medical Branch at Galveston in 1987. She completed her residency at the University of Texas Medical Branch. Dr. Downs specializes in neurology.

Jay S. Epstein, M.D., 400 S. Kingshighway, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Epstein received his baccalaureate degree from Emory University, Atlanta, GA and obtained his medical degree from Emory University School of Medicine, Atlanta, GA in 1983. He completed his residency at Emory University Hospital, Atlanta, GA. Dr. Epstein specializes in pediatrics.

Paula M. Fracasso, M.D., 4960 Audubon Ave., 3rd Floor Wohl Hospital, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Fracasso received her baccalaureate degree from College of the Holy Cross, Worcester, MA and obtained her medical degree from Yale University School of Medicine, New Haven, CT in 1984. She completed her residency at Beth Israel Hospital, Boston, MA. Dr. Fracasso specializes in hematology-oncology.

Thomas J. Francel, M.D., 216 S. Kingshighway, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Francel received his baccalaureate degree from Harvard College, Cambridge, MA and obtained his medical degree from the University of Cincinnati College of Medicine, Cincinnati, OH in 1982. He completed his residency at New England Deaconess Hospital, Boston, MA. Dr. Francel specializes in plastic surgery.

Marjon M. Gillbanks, M.D., 1515 W. Truman Rd., Independence, MO 64080, has become a member of the Missouri State Medical Association. Dr. Gillbanks received her baccalaureate degree from Loyola University, Chicago, IL and obtained her medical degree from Loyola University Stritch School of Medicine, Maywood, IL in 1986. She completed her residency at Lutheran General, Park Ridge, IL. Dr. Gillbanks specializes in internal medicine.

Diana L. Gray, M.D., 216 S. Kingshighway, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Gray received her baccalaureate degree from the University of Illinois, Urbana, IL and obtained her medical degree from the University of Illinois, Urbana, IL in 1981. She completed her residency at Washington University, St. Louis, MO. Dr. Gray specializes in obstetrics/gynecology.

Barry L. Hendrickson, M.D., 222 S. Woods Mill Rd., Chesterfield, MO 63017, has become a member of the Missouri State Medical Association. Dr. Hendrickson received his baccalaureate degree from South Dakota State University and obtained his medical degree from Johns Hopkins University School of Medicine, Baltimore, MD in 1985. He completed his residency at Barnes Hospital/Washington University, St. Louis, MO. Dr. Hendrickson specializes in internal medicine.

Charles B. Huddleston, M.D., 400 S. Kingshighway, Suite 5W24, St. Louis, MO 63110, has become a member of the St. Louis Metropolitan Medical Society. Dr. Huddleston obtained his medical degree from Vanderbilt University School of Medicine, Nashville, TN in 1978. He completed his residency at the Vanderbilt Medical Center, Nashville. Dr. Huddleston specializes in general surgery/thoracic surgery.

Sherry S. Hutchins, M.D., 803 Highway 71 West, Savannah, MO 64485, has become a member of the Buchanan County Medical Society. Dr. Hutchins received her baccalaureate degree from Iowa State University and obtained her medical degree from College of Osteopathic Medicine & Surgery, Des Moines, IA in 1986. She completed her residency at Des Moines General Hospital, Des Moines, IA. Dr. Hutchins specializes in general practice.

Bobby J. Jibben, M.D., 1008 Main, Sikeston, MO 63801, has become a member of the SEMO Medical Society. Dr. Jibben received his baccalaureate degree from Northeast Missouri State University, Kirksville and obtained his medical degree from Kirksville College of Osteopathic Medicine in 1980. Dr. Jibben completed his residency at Normandy Osteopathic Hospitals, St. Louis, MO. Dr. Jibben specializes in emergency medicine.

Timothy R. Jones, M.D., #1 Barnes Hospital Plaza, Suite 17424, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Jones received his baccalaureate degree from the University of Oklahoma, Norman, OK and obtained his medical degree from the University of Oklahoma School of Medicine, Oklahoma City in 1983. He completed his residency at University Hospital, Pittsburgh, PA. Dr. Jones specializes in otolaryngology/plastic surgery.

Randall S. Jotte, M.D., 3635 Vista At Grand Blvd., St. Louis, MO, has become a member of the Missouri State Medical Association. Dr. Jotte received his baccalaureate degree from Vanderbilt University, Nashville, TN and obtained his medical degree from Harvard Medical School, Boston, MA in 1987. He completed his residency at Georgetown University Hospital. Dr. Jotte specializes in emergency medicine.

Ram M. Kakaiya, M.D., 4050 Lindell, St. Louis, MO 63108, has become a member of the Missouri State Medical Association. Dr. Kakaiya received his baccalaureate degree from Bhavans College, Bombay, India and obtained his medical degree from Seth Gordhandas, Sundredas Medical College, Bombay University, Bombay in 1969. He completed his residency at the University of Cincinnati School of Medicine, Cincinnati, OH. Dr. Kakaiya specializes in internal medicine/hematology.

Harold E. Kim, M.D., 510 S. Kingshighway, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Kim received his baccalaureate degree from Loyola University of Chicago, Chicago, IL and obtained his medical degree from Northwestern University Medical School, Chicago in 1987. He completed his residency at Northwestern Memorial Hospital, Chicago, IL. Dr. Kim specializes in radiation oncology.

Soung O. Kwoun, M.D., 11726 St. Charles Rock Rd., Bridgeton, MO 63044, has become a member of the Missouri State Medical Association. Dr. Kwoun received his baccalaureate degree from Chun Nam National University, Kwangju, S. Korea and obtained his medical degree from the College of Medicine, Chun Nam National University, Kwangju, S. Korea in 1962. He completed his residency at Ingham Medical Center, Lansing, MI. Dr. Kwoun specializes in general surgery.

Richard C. Lehman, M.D., 439 S. Kirkwood, Suite 215, St. Louis, MO 63122, has become a member of the Missouri State Medical Association. Dr. Lehman received his baccalaureate degree from the University of Minnesota and obtained his medical degree from the University of Miami School of Medicine, Miami, FL in 1980. He completed his residency at Washington University. Dr. Lehman specializes in orthopedic surgery.

Michael D. Marcus, M.D., 911 So. Brentwood Blvd., St. Louis, MO 63105, has become a member of the St. Louis Metropolitan Medical Society. Dr. Marcus received his baccalaureate degree from Kenyon College, Gambier, OH and obtained his medical degree from the University of Cincinnati College of Medicine, Cincinnati, OH in 1985. He completed his residency at Washington University Barnes Hospital, St. Louis, MO. Dr. Marcus specializes in urology.

James H. McCroy, M.D., 1235 East Cherokee, Springfield, MO 65804, has become a member of the Greene County Medical Society. Dr. McCroy received his baccalaureate degree from Vanderbilt University, Nashville, TN and obtained his medical degree from Tulane University School of Medicine, New Orleans, LA in 1976. He completed his residency at Jacksonville Health Education Program, Jacksonville, FL. Dr. McCroy specializes in pediatrics.

Dace L. Miller, M.D., 1001 E. Primrose, Springfield, MO 65807, has become a member of the Greene County Medical Society. Dr. Miller received his baccalaureate degree from the University of Missouri-Kansas City and obtained his medical degree from the University of Missouri School of Medicine, Kansas City in 1984. He completed his residency at N. C. Memorial Hospital, Chapel Hill. Dr. Miller specializes in internal medicine/gastroenterology.

Tae S. Park, M.D., 400 S. Kingshighway, St. Louis, MO 63110, has become a member of the St. Louis Metropolitan Medical Society. Dr. Park obtained his medical degree from College of Medicine Seoul National University, Seoul, Korea in 1971. He completed his residency at the University of Virginia. Dr. Park specializes in neurosurgery.

William C. Parks, M.D., 1600 E. Broadway, Columbia, MO 65201, has become a member of the Boone County Medical Society. Dr. Parks received his baccalaureate degree from the University of Michigan, Ann Arbor and obtained his medical degree from the University of Michigan Medical School, Ann Arbor in 1977. He completed his residency at the University of Missouri-Columbia, MO. Dr. Parks specializes in family practice.

Joel D. Pavelonis, M.D., 6400 Prospect, Suite 138, Kansas City, MO 64132, has become a member of the Missouri State Medical Association. Dr. Pavelonis received his baccalaureate degree from Ottawa University, Ottawa, KS and obtained his medical degree from the University of Kansas School of Medicine, Kansas City, KS in 1987. He completed his residency at the University of Kansas, Kansas City, KS. Dr. Pavelonis specializes in anesthesiology.

Bruce K. Rubin, M.D., 1425 S. Grand Blvd., St. Louis, MO 63104, has become a member of the Missouri State Medical Association. Dr. Rubin received his baccalaureate degree from Tulane University, New Orleans, LA and obtained his medical degree from Tulane University School of Medicine, New Orleans in 1979. He completed his residency at Tulane Medical Center Hospital. Dr. Rubin specializes in pediatrics.

Anthony Sabatino, M.D., 6400 Prospect, Kansas City, MO 64132, has become a member of the Missouri State Medical Association. Dr. Sabatino received his baccalaureate degree from Creighton University, Omaha, NB and obtained his medical degree from Creighton University School of Medicine, Omaha, NB in 1987. He completed his residency at State University of New York at Stoney Brook School of Medicine, Stony Brook, NY. Dr. Sabatino specializes in anesthesiology.

Frank J. Schlehr, M.D., 2301 Holmes, Kansas city, MO 64108, has become a member of the Missouri State Medical Association. Dr. Schlehr received his baccalaureate degree from the University of Notre Dame, Notre Dame, IN and obtained his medical degree from Vanderbilt University School of Medicine, Nashville, TN in 1984. He completed his residency at the University of Kansas Hospital, Kansas City, KS. Dr. Schlehr specializes in orthopaedics.

Daniel Scodary, M.D., 12255 DePaul Dr., Suite 830, Bridgeton, MO 63044, has become a member of the St. Louis Metropolitan Medical Society. Dr. Scodary obtained his medical degree from St. Louis University School of Medicine, St. Louis, MO in 1985. He completed his residency at the University of Cincinnati, Cincinnati, OH. Dr. Scodary specializes in neurosurgery.

Larry A. Scroggins, M.D., 1600 East Broadway, Columbia, MO 65201, has become a member of the Boone County Medical Society. Dr. Scroggins received his baccalaureate degree from the University of Arkansas, Fayetteville and obtained his medical degree from the University of Arkansas School ot Medicine, Little Rock in 1988. He completed his residency at the University of Missouri-Columbia, MO. Dr. Scroggins specializes in family practice.

Steven M. Silverstein, M.D., 17500 Eyes of Missouri Medical Ctr. Pkwy., Suite 7, Independence, MO 64057, has become a member of the Missouri State Medical Association. Dr. Silverstein received his baccalaureate degree from Emory University, Atlanta, GA and obtained his medical degree from the University of Oklahoma College of Medicine, Oklahoma City in 1986. He completed his residency at Tufts New England Medical Center, Boston, MA. Dr. Silverstein specializes in ophthalmology.

Joseph P. Slimack, M.D., 3015 N. Ballas Rd., Suite 35, St. Louis, MO 63131, has become a member of the Missouri State Medical Association. Dr. Slimack received his baccalaureate degree from Eureka College, Eureka, IL and obtained his medical degree from the University of Illinois at Chicago Health Sciences Center, Chicago in 1984. He completed his residency at Naval Hospital, Oakland, CA. Dr. Slimack specializes in anesthesiology.

Toni R. Smith, M.D., 1104 N. Baltimore, Kirksville, MO 63501, has become a member of the North Central Medical Society. Dr. Smith received her baccalaureate degree from Northeast Missouri State University, Kirksville and obtained her medical degree from Kirksville college of Osteopathic Medicine, Kirksville, MO in 1979. She completed her residency at Kirksville Osteopathic Medical Center. Dr. Smith specializes in anesthesiology.

Michael E. Somers, M.D., 2700 Clay Edward Dr., North Kansas City, MO, has become a member of the Missouri State Medical Association. Dr. Somers received his baccalaureate degree from Brown University, Providence, RI and obtained his medical degree from Brown University of Biological-Medical Sciences, Providence, RI in 1984. He completed his residency at Yale Eye Center, New Haven, CT. Dr. Somers specializes in ophthalmology.

Carol J. Soucie, M.D., 1465 S. Grand Blvd., St. Louis, MO 63104, has become a member of the Missouri State Medical Association. Dr. Soucie received her baccalaureate degree from Colgate University, Hamilton, NY and obtained her medical degree from St. Louis University School of

Medicine, St. Louis, MO in 1988. She completed her residency at St. Louis University Hospital, St. Louis, MO. Dr. Soucie specializes in pediatrics.

Carmen K. Steigman, M.D., 1465 S. Grand Blvd., St. Louis, MO 63104, has become a member of the Missouri State Medical Association. Dr. Steigman received her baccalaureate degree from Northeast Louisiana University, Monroe LA and obtained her medical degree from Louisiana State University School of Medicine, New Orleans, LA in 1983. She completed her residency at Fairfax Hospital, Falls Church, VA. Dr. Steigman specializes in pathology.

Timothy E. Stepp, M.D., 6675 Holmes, Suite 420, Kansas City, MO 64131, has become a member of the Missouri State Medical Association. Dr. Stepp received his baccalaureate degree from Baylor University, Waco, TX and obtained his medical degree from the University of Kansas School of Medicine, Kansas City in 1985. He completed his residency at University Hospital, Kansas City, KS. Dr. Stepp specializes in neurosurgery.

Eric J. Sutphen, M.D., 6150 Oakland Ave., St. Louis, MO 63139, has become a member of the Missouri State Medical Association. Dr. Sutphen received his baccalaureate degree from the University of California, Los Angeles, CA and obtained his medical degree from St. Louis University School of Medicine, St. Louis, MO in 1987. He completed his residency at St. Mary's Hospital, San Francisco, CA. Dr. Sutphen specializes in radiation oncology.

Celeste A. Tarantino, M.D., 24th & Gillham, Kansas City, MO 64108, has become a member of the Missouri State Medical Association. Dr. Tarantino received her baccalaureate degree from the University of Missouri, Columbia and obtained her medical degree from the University of Kansas School of Medicine, Kansas City in 1988. She completed her residency at Children's Mercy Hospital, Kansas City, MO. Dr. Tarantino specializes in pediatrics.

Kara L. Tate, M.D., 9332 Harrison, Kansas City, MO 64131, has become a member of the Missouri State Medical Association. Dr. Tate received her baccalaureate and medical degrees from the University of Missouri School of Medicine, Kansas City, MO in 1987. She completed her residency at St. Luke's Hospital, Kansas City, MO. Dr. Tate specializes in anesthesiology.

Gene Tenorio, M.D., 1000 East Primrose, Springfield, MO 65807, has become a member of the Greene County Medical Society. Dr. Tenorio received his baccalaureate degree from Texas Tech University, Lubbock, TX and obtained his medical degree from Texas Tech University School of Medicine, Lubbock in 1984. He completed his residency at Texas Tech, Amarillo. Dr. Tenorio specializes in pediatric neurology.

Randall E. Treadwell, M.D., 4444 Forest Park, St. Louis, MO 63108, has become a member of the St. Louis Metropolitan Medical Society. Dr. Treadwell obtained his medical degree from the University of Texas Southwestern Medical School, Dallas in 1978. He completed his residency at John Peter Smith Hospital, Ft. Worth, TX. Dr. Treadwell specializes in family practice.

Denise R. Trowbridge, M.D., 1309 E. 154th, Olathe, KS 66062, has become a member of the Missouri State Medical Association. Dr. Trowbridge received her baccalaureate degree from Baker University, Baldwin, KS and obtained her medical degree from the University of Kansas School of Medicine, Kansas City in 1987. She completed her residency at the University of Kansas. Dr. Trowbridge specializes in radiation oncology.

Thomas D. Tyler, M.D., 2300 Old Spanish Trail, #2055, Houston, TX 77054, has become a member of the Lafayette-Ray Medical Society. Dr. Tyler received his baccalaureate degree from the University of Kansas, Lawrence, KS and obtained his medical degree from the University of Texas Medical School, Houston in 1986. He completed his residency at the University of Texas Medical School. Dr. Tyler specializes in ophthalmology.

Teresa J. Vietti, M.D., 400 S. Kingshighway, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Vietti received her baccalaureate degree from Rice University, Houston, TX and obtained her medical degree from Baylor University College of Medicine, Houston, TX in 1953. She completed her residency at Children's Hospital, Detroit, MI. Dr. Vietti specializes in pediatric hematology/oncology.

Thuy-Lieu T. Vo, M.D., 216 S. Kingshighway, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Vo received her baccalaureate degree from the University of South Dakota, Vermillion, SD and obtained her medical degree from Washington University School of Medicine, St. Louis, MO in 1985. She completed her residency at Washington University School of Medicine, St. Louis. Dr. Vo specializes in clinical/anatomical pathology.

Dennis G. Vollmer, M.D., 660 S. Euclid, St. Louis, MO 63110, has become a member of the St. Louis Metropolitan Medical Society. Dr. Vollmer obtained his medical degree from the University of Texas Medical School, San Antonio in 1979. He completed his residency at the University of Virginia. Dr. Vollmer specializes in neurosurgery.

Harry L. Wadsworth, M.D., 3009 N. Ballas Rd., Suite 100, St. Louis, MO 63131, has become a member of the Missouri State Medical Association. Dr. Wadsworth received his baccalaureate degree from Texas Tech University, Lubbock and obtained his medical degree from Texas Tech University School of Medicine, Lubbock in 1983. He completed his residency at Texas Tech University, Lubbock. Dr. Wadsworth specializes in internal medicine/endocrinology.

Mark E. Weaver, M.D., 1900 S. National, Springfield, MO 65804, has become a member of the Greene County Medical Society. Dr. Weaver received his baccalaureate degree from the University of Arkansas and obtained his medical degree from the University of Arkansas School of Medicine, Little Rock in 1981. He completed his residency at Barnes Hospital, St. Louis, MO. Dr. Weaver specializes in anesthesiology.

Charlie B. Williams, Jr., M.D., 1115 Alaska Ave., Suite 112, West Plains, MO 65775, has become a member of the South Central Medical Society. Dr. Williams, Jr. received his baccalaureate degree from Emory University, Atlanta, GA and obtained his medical degree from Emory University School of Medicine, Atlanta, GA in 1966. He completed his residency at Emory University Affiliated Hospitals, Atlanta, GA. Dr. Williams specializes in surgery.

Ronald M. Wyatt, M.D., 5701 Delmar, St. Louis, MO 63112, has become a member of the Missouri State Medical Association. Dr. Wyatt received his baccalaureate degree from the University of Alabama, Birmingham and obtained his medical degree from the University of Alabama School of Medicine, Birmingham in 1985. He completed his residency at St. Louis University Medical Center, St. Louis, MO. Dr. Wyatt specializes in internal medicine.

RESIDENTS

Luis J. Anglo, M.D. - urology
Salma S. Babar, M.D. - emergency medicine
Sanjay T. Bhat, M.D. - internal medicine
Jean E. Birmingham, M.D. - pediatrics
Saad R. Bitar, M.D. - neurology
Jeffrey L. Boesch, M.D. - internal medicine
Sara A. Bohn, D.O. - family practice
David M. Brown, M.D. - general surgery
Lilibeth M. Cayabyab-Loe, M.D. - internal medicine
Lawrence L. Creswell, M.D. - general surgery
Maria C. Dumadag-Sabio, M.D. - internal medicine
Robert E. Garola, M.D. - pathology
Brett D. Hosley, D.O. - neurology
Wit A. Jamry, M.D. - internal medicine
Daniel Keleti, M.D. - radiation oncology
Jeseph B. Meyer, M.D. - anesthesiology
Jonathan W. Mink, M.D., Ph.D. - pediatric neurology
Gary R. Peplinski, M.D. - general surgery
Juan J. Pineda, M.D. - internal medicine
Raymunde E. Ramirez, M.D. - internal medicine
Joseph P. Romeo, M.D. - otolaryngology
Debabrata Saha, M.D. - psychiatry
Lawrence A. Scariano, M.D. - internal medicine
Adam J. Sky, M.D. - geriatric psychiatry
David D. Springer, M.D. - pathology
Ruanne M. Stamps, M.D. - geriatrics
Tracy L. Stevens, M.D. - internal medicine
Brettly J. Teague, M.D. - ophthalmology
Tony N. Tsen, M.D. - internal medicine
Cleo Tsolakoglou, M.D. - obstetrics/gynecology
Ozemenam, I. Uketui, M.D. - family practice
Erik J. Wait, M.D. - obstetrics/gynecology
William P. Wright, Jr., M.D. - internal medicine/cardiology

STUDENT SECTION

St. Louis University

Michele Brother
Mark J. McDonald

University of Missouri-Columbia

Kevin Jones
Elizabeth N. Lumpkin
Joseph Reuben

University of Missouri-Kansas City

Komal B. Chaundhary
Robert B. Gerber
Suzanne Hoover
Darshan Shah
Donna Taulbee
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Medical School News

ST. LOUIS UNIVERSITY

Maurice Green, Ph.D., professor and chairman of the Institute for Molecular Virology (IMV), St. Louis University School of Medicine, has been awarded a five-year, $650,385 grant from the National Cancer Institute (NCI). The grant was awarded following a specific request for applications from the NCI for new approaches aimed at understanding how certain cancer-causing viruses trigger the transformation of normal cells to cancerous ones.

Dr. Green and his colleagues at the IMV are studying how viruses modify growth regulatory processes in cells. Using the human adenovirus as a model, the team has identified and chemically synthesized portions, or domains, of a cancer-causing gene called the E1A oncogene. These domains can, at times, repress cellular activity or activate cells to divide.

The research may identify critical signal pathways and cellular proteins that regulate cell growth and, thus, protect against malignant growth. Paul M. Loewenstein, assistant research professor, is co-investigator of this study.

Dr. Green, Ph.D., also has been awarded a prestigious four-week fellowship from the Japanese Society for the Promotion of Science. The fellowship allows Dr. Green to spend 28 days in numerous medical and cancer research facilities in Japan lecturing on cancer and AIDS related research. He will also give a historical perspective of research on tumor viruses (viruses that cause cancer in animals).

Dr. Green will lecture at 18 different universities and cancer research centers.

Bernard Chaltman, M.D., professor of internal medicine and director of the division of cardiology, St. Louis University School of Medicine, has been given a $228,000 grant from the National Institutes of Health for the "Rest and Exercise Core Laboratory for Asymptomatic Cardiac Ischemia (ACIP) Study."

The international study will help determine the best way of treating patients with asymptomatic cardiac ischemia. The grant allows St. Louis University School of Medicine to serve as the core ECG laboratory for the study. The ECGs will be coming from eight medical facilities in the United States, including St. Louis University, plus two facilities in Canada and one in England.

Chaltman also has received two grants totaling $86,944 from CIBA-GEIGY to participate in the TIMI 5 Open Label study.

The study is designed to determine whether the drug r-hirudin, is more effective than Heparin, another anticoagulant, in thinning the blood and preventing blood clots from forming in the arteries.

A portion of the grant, $62,300, will be used for the clinical trials of r-hirudin. The other $24,544 will pay for analysis of electrocardiograms.

Clifford Bellone, Ph.D., professor of microbiology at St. Louis University School of Medicine has received a $200,000 grant to participate in the G.D. Searle & Co. Arthritis and Prostaglandins Research Challenge. The challenge is a $10 million effort to discover what role prostaglandins play in arthritis and other related immune disorders.

Dr. Bellone is one of 61 scientists in the United States and Canada participating in the challenge. His research will focus on a hormone-like substance known as interleukin-1.

When interleukin-1 is overproduced for prolonged periods of time it leads to chronic inflammation. This is the case with arthritis patients. Eventually, the chronic inflammation wears down the bone and damages cartilage.

Dr. Bellone's research will work toward the identification of proteins that activate the gene that produces interleukin-1. If researchers can determine the components responsible for turning on the gene, they may be able to control its production.

UNIVERSITY OF MISSOURI - COLUMBIA

James T. Barrett, Ph.D., professor of molecular microbiology and immunology, recently returned from a trip to Thailand, where he presented a seminar series on topics in immunology at Prince of Songkhla University in Hat Yai.

Sheldon Braun, M.D., professor and director of pulmonary, critical care and environmental medicine, has received a five-year, $375,000 National Institutes of Health Preventive Pulmonary Academic Award for curriculum development.

Thomas Burns, M.D., professor and director of endocrinology, metabolism and diabetes, recently was presented the Missouri Society of Internal Medicine's 1991 Special Recognition Award. The honor was given during the annual join meeting of the

Missouri chapter of the American College of Physicians and the Missouri Society of Internal Medicine at the Lake of the Ozarks. Burns, who also is director of MU's Cosmopolitan International Diabetes Center, received the award for his extensive contributions to the ACP, MSIM and Missouri medicine. He has been a School of Medicine faculty member since 1955.

Maria L. Evans, M.D., a resident physician in the department of pathology, has been appointed to the Residents and Juniors Committee of the College of American Pathologists. The only resident on the committee, she will serve a one-year term.

Ramareddy V. Guntaka, Ph.D., professor of molecular microbiology and immunology, recently was awarded a three-year, $283,764 grant from the U.S. Environmental Protection Agency to study toxic chemicals found in drinking water. In humans, the liver eliminates many of these substances, but some of them have been found to cause liver tumors in experimental animals. Guntaka is trying to find the genes that are involved in causing such tumors.

Mark Hannink, Ph.D., assistant professor of biochemistry, has received a two-year grant from the March of Dimes. He is studying how the V-rel protein, a structurally altered form of a normal cellular protein, can cause cells to become cancerous.

Robert W. Hoffman, D.O., assistant professor of immunology and rheumatology, has received two new research awards to study the genetic causes of connective tissue diseases and autoimmunity. The National Institutes of Health recently awarded him a five-year, $375,000 grant, and he also received a three-year, $154,000 grant from the U.S. Department of Veterans Affairs.

Thomas W. Hurley, M.D., associate professor of child health and physiology, has accepted an invitation from the National Institutes of Health to serve a four-year term as a member of its Oral Biology and Medicine Study Section of the Division of Research Grants. Members are selected on the basis of demonstrated competence and achievement in their scientific discipline as evidenced by the quality of research accomplishments, publications in scientific journals and other significant scientific activities, achievements and honors. Study sections review grant applications submitted to the NIH, make recommendations on these applications to the appropriate NIH national advisory council or board and survey the status of research in their fields of science.

Michael C. Perry, M.D., professor of hematology and oncology, has completed *The Chemotherapy Source Book*, which is billed as "the one complete resource for chemotherapy." The 1,100-page book was published in October by Williams & Wilkins of Baltimore, Md., and contains 61 chapters with material from 78 world-class authorities on chemotherapy.

Lisa J. Pierce, M.D., assistant professor of family and community medicine, has been awarded a $1,500 Parke-Davis Teacher Development Award. One of 12 winners in 1991, she was selected from 101 applicants. The awards are given to individuals completing their residencies who plan to enter academic medicine. Pierce, who completed her residency at MU in July 1991, participates in patient care, teaching and research activities through the department's satellite practice in Fulton.

P. Kevin Rudeen, Ph.D., associate professor of anatomy and neurobiology, has received continued research funding of $290,250 for 1991-92 to study fetal alcohol syndrome. Funded by the National Institute of Alcohol Abuse and Alcoholism, he is looking at how alcohol affects the development and differentiation of the central nervous system in the fetus.

Willis K. Samson, Ph.D., professor of anatomy and neurobiology, has been awarded a three-year, $132,000 grant from the American Heart Association. He is trying to determine the role of a newly discovered hormone, endothelin-3, in the brain and how it may control cardiovascular function. Samson was one of the first researchers to demonstrate endothelin-3's presence in the brain and was the first to show that it is active in the brain.

Daniel S. Smith, M.D., assistant professor of pathology, recently received a two-year, $135,000 renewal grant to study the enzymatic modification of red-cell antigens. His goal is to produce universally transfusable red blood cells.

Twelve mid-Missouri counties are represented by a new regional advisory board appointed for the MU Children's Center. The board includes local and regional leaders whose communities are served by the center. Members will help the Children's Center meet the needs of the 1990s through community outreach, advocacy and fund raising.

The members are: Muriel Battle, Tom Gray, Robert Harris, M.D., Nancy James, Gus Lehr, Kay Lindner, David Moore, Joe Moseley, Jodie Needy, Wayne Sells, Dan Scotten and Russell Thompson, all of Columbia; William Backer and George Oestreich of Fulton; Jim Carl of Centralia; Anne Elsberry of Kansas City; Carolyn Fleming of Moberly; Doug Groesbeck of Eldon; Judy Hackman of Jefferson City; Donna Huston of Marshall; Robert Keil of California; Raymond Lansford of Lake Ozark; Bob L. McDonald of Sedalia; Pam May of Camdenton; Audry Walton of Versailles; actress Dawn Wells of Toluca Lake, California; and Tim Williams of Mexico.

University of Missouri Kansas City

A new two-way video capability is enhancing the educcational exchange between the School of Medicine and Saint Luke's Hospital.

Two-way, interactive communication is possible via American Cablevision's I-Net, broadcasting from St. Luke's to a video-equipped theater at the Medical School. The technology enables physicians and students at both locations to communicate with a lecturer or panelist at either location.

The two-way video is a particular asset that resulted from the merger two years ago of the Medical School and Saint Luke's educational programs.

The departments of anesthesioloby and cardiology at Saint Luke's and Truman Medical Center, UMKC's promary teaching hospital, are the first to use the two-way video. Many of the combined residency programs created by the affiliation will take advantage of this new teaching tool.

Ticket donations and bids from the first UMKC School of Medicine Benefit Auction October 18 brought in more than $12,000 to establish a Scholarship and Student Aid Fund.

More than 100 people, including administrators, faculty, students and friends of the medical school participated in the auction that was held at the Savoy Hotel in Kansas City.

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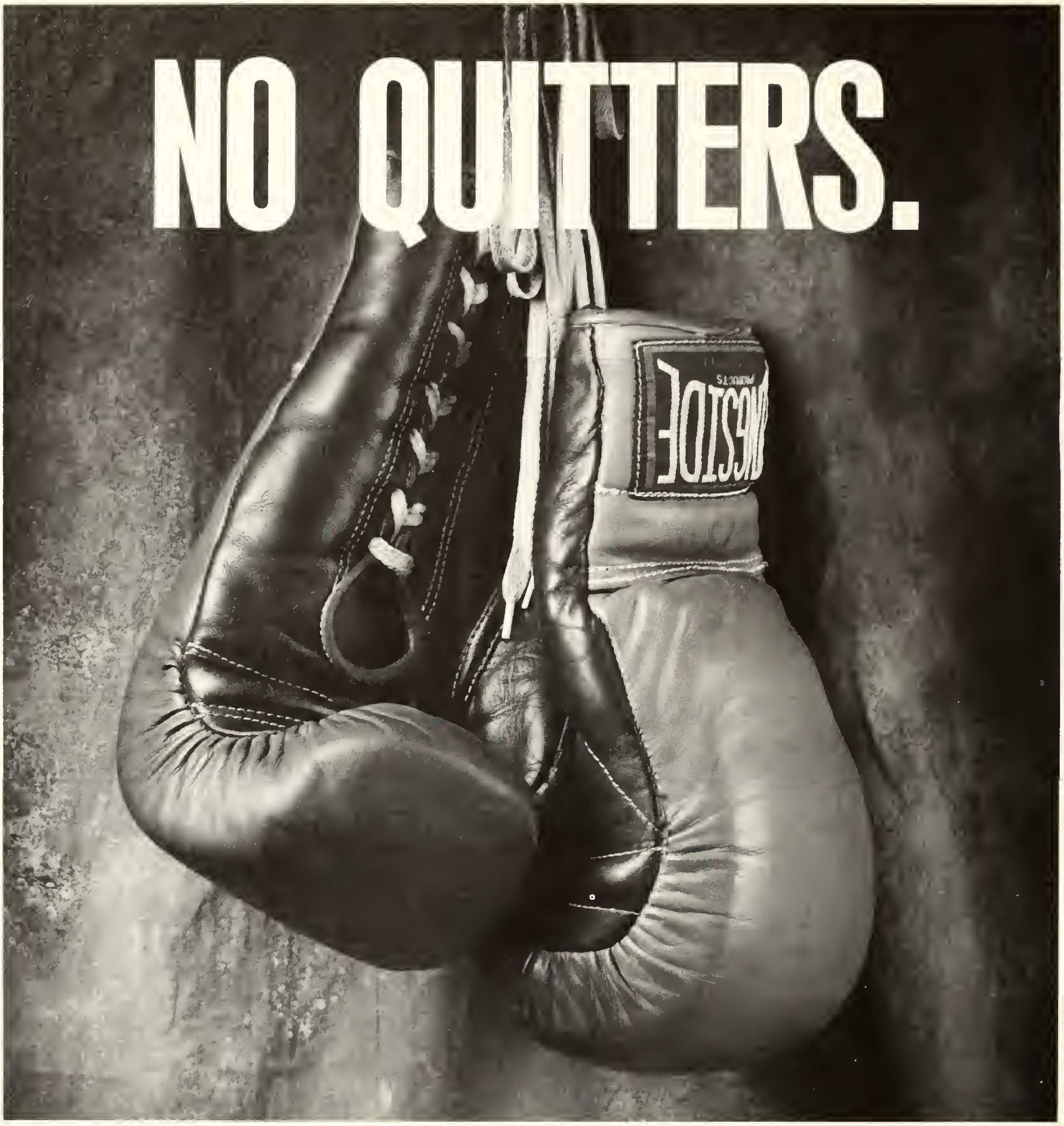

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JANUARY

7 - 8 **Transesophageal Echocardiography** - Location: Saint Louis University medical center. Contact: CME Office, St. Louis University Medical Center, 1402 South Grand Blvd., St. Louis, Missouri 63104. (800) 553-2712.

11 - 12 **Advanced Cardiac Life support Provider Course** - Location: Margaret McCormick Doisy Learning Resources Center. St. Louis University Medical Center. Contact: CME Office, SLU-Medical Center, 1402 South Grand Blvd., St. Louis, MO 63104. (800)553-2712.

13 **Hypercholesterolemia In Women** - Location: Lutheran Medical Center, First Floor Conference Room, OB/GYN Departmental Meeting, 2639 Miami Street, St. Louis, MO 63118.

13 **City-Wide Radiology Conference**- Location: St. Louis University. Contact: R. Gilbert Jost, M.D., Chief of Diagnostic Radiology, Mallinckrodt Institute of Radiology, 510 South Kingshighway, St. Louis, MO 63110. 314/362-7130.

16 - 18 **American Academy of Pain Medicine's 1992 Annual Conference** - Location: Registry Hotel, Scottsdale, AZ. Contact: Carol Endicott, American Academy of Pain Medicine, 5700 Old Orchard Road, First Floor, Skokie, IL 60077-1024. 708/966-9510.

20 - 24 **Medicolegal Death Investigator Training Course** - Location: Margaret McCormick Doisy Learning Resources Center. Contact: CME Office, SLU Medical Center, 1412 Grand Blvd., St. Louis, MO 63104. 314/577-8167.

29 **Updates in Surgery,** The Fourth annual Joseph E. Flynn Lecture Series - Location: von Gontard conference Center, St. John's Mercy Medical Center, 615 South New Ballas Road, St. Louis, MO 63141. Contact: Judith L. O'Guin. 314/569-6290.

FEBRUARY

3 - 6 **Practical Anatomy & Surgical Technique Workshop Temporal Bone Surgical Dissection** - Hand-on workshop. Location: St. Louis Metropolitan Medical Society Building. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

7 **Otology Update** - Location: St. John's Mercy Medical Center. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

7 **Interventional Radiology** - Location: Research Medical Center. Contact: The Ed Bixby Institute for Postgraduate Medical Education, 2316 East Meyer Boulevard, Kansas City, Missouri 64132. Missouri: (800) 892-5771, ext. 4306. Bordering states: (800) 821-5140, ext. 4306.

7 - 8 **Midwinter Laser Course & Fluorescein Angiography Workshop** - Location: Hyatt Regency Hotel, St. Louis, MO. Contact Leigh Dundon, Suite 17413, East Pavilion, One Barnes Hospital Plaza, St. Louis, MO 63110. 314/367-1181.

10 **Evaluation of Myocardial Viability** - Location: Mallinckrodt Institute of Radiology, St. Louis, Mo. Contact: R. Gilbert Jost, M.D., chief of Diagnostic Radiology, Mallinckrodt Institute, 510 South Kingshighway, St. Louis, MO 63110. 314/362-7130.

11 - 12 **Introduction to Doppler Echocardiography** - Location:Saint Louis University Medical Center. Contact: CME Office, SLU, 800/553-2712 or 577-8167.

19 - 21 **Twelfth Annual Conference on Peritoneal Dialysis** - Location: Seattle Convention Center, Seattle, Washington. Contact Barry Kling, UMC, MA215 Health Sciences Center. 314/882-4105.

MARCH

3 - 4 **Transesophageal Echocardiography Course** - Location: Saint Louis University Medical Center. Contact: CME Office, SLU, 1402 S. Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 **Current Perspectives in Cancer Therapy, The Multi-modal Approach** - Location: The Ritz-Carlton Hotel, St. Louis, MO. Contact: CME Office, SLU , 1402 South Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 - 7 **Advanced Trauma Life Support Provider** - Location: Margaret McCormick Doisy Learning Resources Center. Contact: CME Office, SLU, 1402 South Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 - 8 **Family Practice Update** - Location: UMC School of Medicine, Columbia, MO. Contact: Barry Kling, CME Office, MA215 Health Sciences Center. 314/882-4105.

9 **CT of the Lung Parenchyma** - Location: Mallinckrodt Institute of Radiology. Contact: R. Gilbert Jost, M.D., Mallinckrodt Institute of Radiology, 510 S. Kingshighway Boulevard, St. Louis, MO 63110. 314/362-7130.

13 **Orbit and Eyelid Surgery** - Location: Margaret McCormick Doisy Learning Resources Center, Saint Louis University. Contact: CME Office, SLU, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

27 **Tenth Annual Geriatric Symposium** - Location: Breckenridge Frontenac, St. Louis, MO. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

APRIL

4 **Liver Transplantation for the Primary Care Physician**- Location: Margaret McCormick Doisy Learning Resources Center, St. Louis University. Contact: CME Office, SLU, 1412 South Grand blvd., St. Louis, MO 63104. 314/577-8167.

13 **City-Wide Radiology Conference** - Location: Mallinckrodt Institute of Radiology, Washington University Medical Center. Contact: R. Gilbert Jost, M.D., 510 South Kingshighway Blvd. St. Louis, MO 63110. 314/362-7130.

MAY

7 - 10 **Emergency Department Legal Environment** - Location: St. Petersburg Beach, Florida. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

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In addition, in view of The Copyright Revision Act of 1976, effective Jan. 1, 1978, transmittal letters to the editor should contain the following language: "In consideration of the Missouri State Medical Association's taking action in reviewing and editing my submission, the author(s) undersigned hereby transfers, assigns or otherwise conveys all copyright ownership to the MSMA in the event that such work is published by the MSMA." We regret that transmittal letters not containing the foregoing language signed by all authors of the submission will necessitate delay in review of the manuscript. Please include the title of the article in the transmittal letter.

Manuscripts

Manuscripts should be typewritten, double-spaced, and the original with one copy submitted. Retain another copy for proofreading. Please number the pages of your manuscript. Manuscripts are not returned. Hospital and University appointments of the author(s) should accompany the manuscript. A synopsis-abstract of no more than 75 words should accompany the manuscript. Manuscripts may be submitted on 5¼ or 3½ floppy disks using WordPerfect 5.1 or below.

References

References should be cited in numerical order in the text and arranged at the end of the article in the same order in which they are cited. The reference should give name of author, title of article, name of periodical, volume number, initial page number and year. Authors are responsible for reference accuracy.

Illustrations

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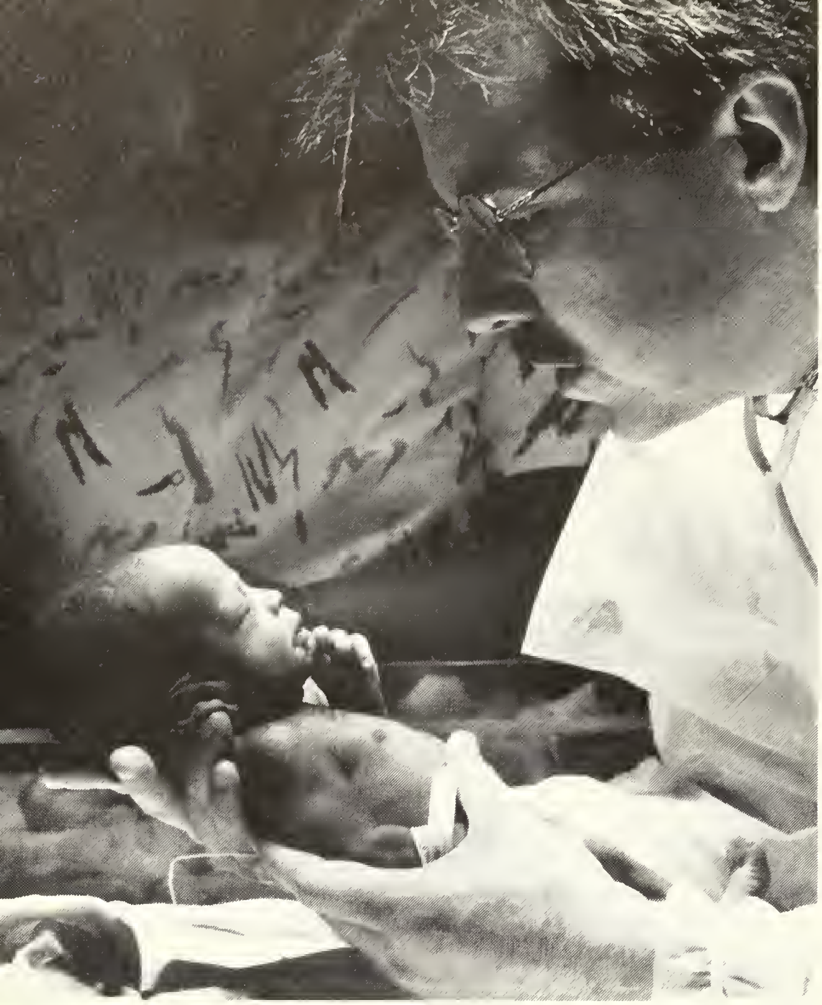

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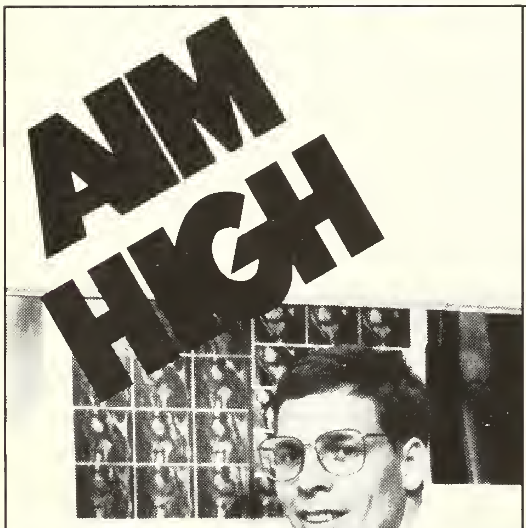

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AIR FORCE

Disciplinary Actions

The following is a listing of final disciplinary actions taken by the State Board of Registration for the Healing Arts. The listing includes actions finalized through May 17, 1991. This information made available to the pubic under the provisions of Section 334.101 RSMo. Names underlined indicates members of the Missouri State Medical Association.

■ **MYRAL C. COATNEY, D.O.**, Kansas City, MO. Delegated professional responsibilities to his assistant who was not qualified to perform them; Dr. Coatney improperly prescribed and overprescribed to numerous patients; and he failed to cooperate during the course of the Board's investigation.
Violation: Section 334.100.2(4c)(4d)(4h)(41)(6)(10) RSMo Supp. 1989
Board Action: Two (2) month suspension to begin on 4/19/91

■ **PATRICK M. COYNE, M.D.**, Florissant, MO. Violated the Missouri Board's Amphetamine Rule which instructs the doctor not to prescribe more than a 30-day supply of amphetamine agents per prescription. Dr. Coyne authorized refills without seeing the patient.
Violation: Section 334.100.2(6) RSMo Supp. 1990
Board Action: Agreement -- surrendered all anorectic controlled substance authority and shall, within one year, complete a pharmacology course acceptable to the Board. Effective 7/23/91

■ **WALTER J. KUTRYB, M.D.**, St. Louis, MO. Limitation of controlled substance registration as a result of overprescribing, personal impairment and prescribing of controlled drugs to wife with no medical records documentation.
Violation: Section 334.100.2(1)(4h((13)(22) RSMo Supp. 1989/1990
Board Action: License voluntarily surrendered, reinstated on a five (5) year probation. Effective 5/20/91

■ **MICHELLE A. MARTIN, M.D.**, St. Louis, MO.
Board Action: Termination of probation. Effective 7/12/91

■ **RICHARD E. MOORE, M.D.**, Cape Girardeau, Missouri. Personal impairment.
Violation: Section 334.100.2(1)(4h)(5)(13)(22)(24) RSMo Supp. 1989
Board Action: License voluntarily surrendered, reinstated on a five (5) year probation. Effective 7/16/91

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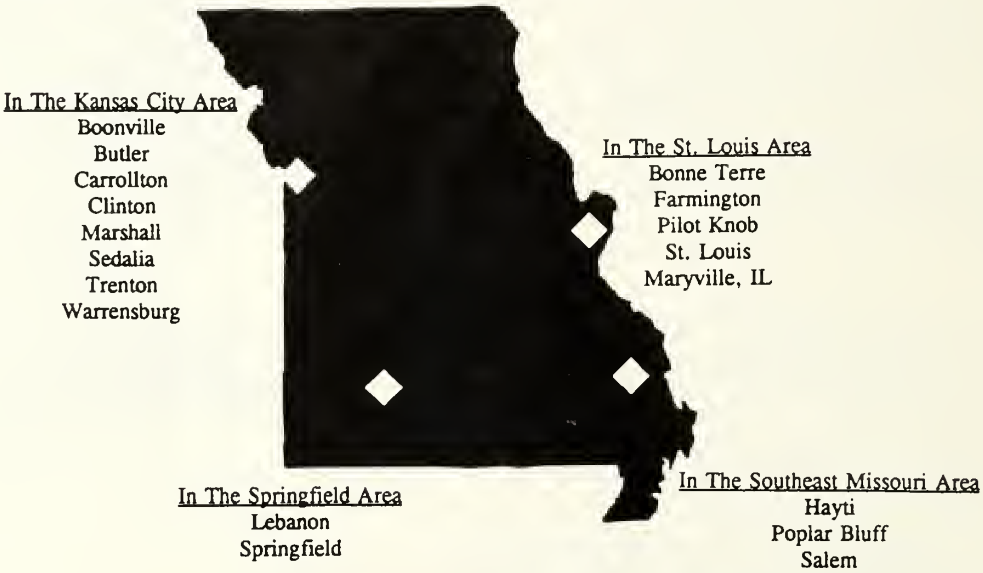
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Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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Missouri Medicine

Journal of the Missouri State Medical Association

February 1992

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- *Final Medicare and Medicaid Anti-Kickback Safe Harbor Regulations*
- *Thanatology: A New Clinical Specialty to Help Confront Death*
- *Invasive Pulmonary Aspergillosis Complicated by Nocardiosis in a Patient with Agnogenic Myeloid Metaplasia*
- *Seat Belt Usage by Trauma Center Personnel*
- *Recent Trends in the Selection and Fitting of Hearing Aids*

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Al Agnew, a native of the eastern Missouri Ozarks, produced "*Vilander Bluff*" to show some of the beauty that would have been lost to future generations if the Corps of Engineers had succeeded in building dams along the Meramec during the 1970s. Construction of the dams was hotly debated and finally voted down in 1981.

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MONETAMED PROFILE

THOMAS P. DOYLE, M.D.

HOME: Springfield, Missouri.

AGE: 36.

SPECIALTY: Orthopedic Surgery.

MEDICAL COLLEGE: University of Missouri, 1978

PRACTICE: "I recently purchased an established but declining sole practitioner office from a retiring physician. It's a risk, but they say that great deeds are usually wrought at great risk."

TYPICAL DAY: "Let the horses out of the corral at dawn and maybe even go for a ride. Then, it's surgery if scheduled and a day of torn ligaments and sprained ankles. The evening finds me on the sidelines of one of our high school sports teams."

HOBBIES: Horseback riding and golf. "If I combined activities, would it be polo?"

LAST BOOK READ: Missouri: Images of Nature, photographed by Charles Gurche.

LATEST ACCOMPLISHMENT: "Testing a protective knee pad I'm developing for use by football players."

QUOTE: "Physicians are being deterred from trying new procedures and therapies in patient care. We need to cut the chains and encourage individual innovation."

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Missouri Medicine (ISSN 0026-6620) is published monthly by the Missouri State Medical Association, P.O. Box 1028, 113 Madison St., Jefferson City, MO 65102.
Subscription rates: $20 per year.
Second class postage paid at Jefferson City, Missouri and at additional mailing offices.
Postmaster: Send address changes to Missouri State Medical Association, c/o Membership Dept., P.O. Box 1028, Jefferson City, MO 65102.

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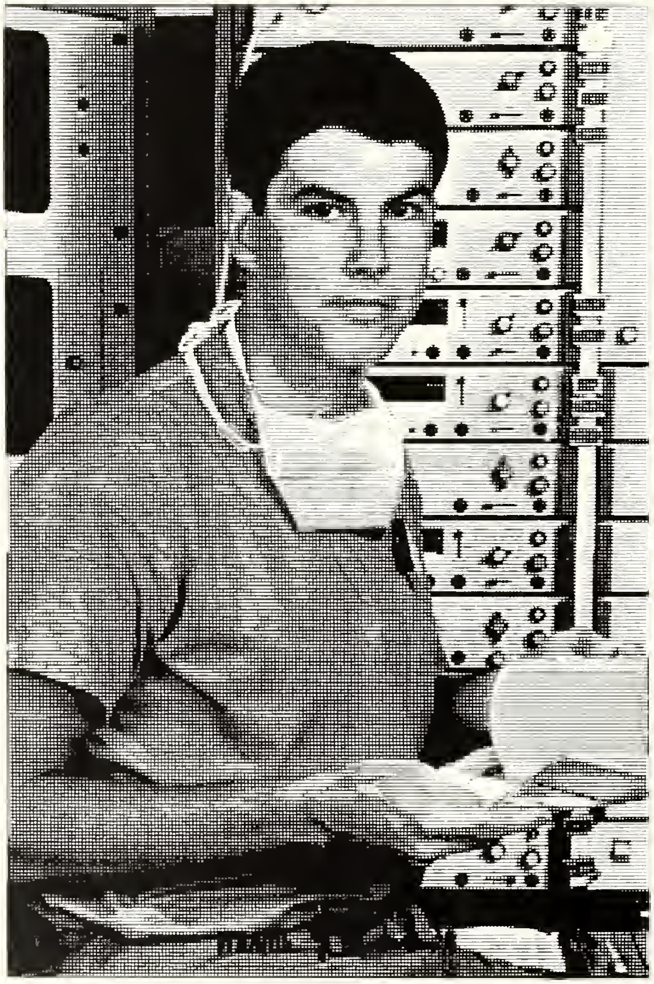

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EDITOR'S DESK

by
Donald G. Sessions, M.D.
Editor

Will We Be All Right ?

Will we be all right? That's a good question to ask these days. It is very appropriate at times to stop what we are doing and observe what is going on around us. What is your mood and what in fact is the corporate mood? Mood has been described as an automatic assessment of our possibilities for the future. Utilizing that definition the mood of corporate medicine is extremely low. If you are privileged enough to practice medicine currently you are well aware of the many intrusions that seem to interfere with your ability to care for your patients. It is impossible to keep up with the body of knowledge. It is not possible to stay on the leading edge of the new technology. Every time we turn around there is a new form to fill out, a stack of papers to sign, charts to dictate, charges to code and re-code, urgent phone calls to return, patients to see, committee meetings to attend, complaints to listen to, mail to go through, new regulations to review, results to check on, patients to report results to, referring physicians to contact, pre-authorizations to get, informed consents to obtain, and on and on. Literally every action we take involves adding to the time that we spend on the job. If we are smart enough to teach or train others to do these jobs then that takes time. And this all takes place historically at a time when it appears permanently impossible to be reimbursed for this seemingly endless list of tasks that must be done. Also, if we are unable to "heal" our patients or handle their concerns in a manner that meets their expectations then we are eligible to be attacked with complaints and allegations and charges and even law suits.

Will we be all right? When we observe what is happening to health care in our country it is clear that something must be done to handle the problems in two areas: controlling the cost in the system and opening the system to a large underserved part of the population. It's hard to ignore twenty to thirty million people. As we brag about having the best medicine in the world it's impossible to continue to ignore our abysmal rate of infant mortality. It is much easier to blame it on poverty than it is for us to take the responsibility as part of a seemingly uncaring health system. As a national corporate entity, physicians have an opportunity to take a moral stand in their willingness and their duty to care for their constituency - their patients.

Will we be all right? That's a great question when we try to figure out who is going to pay for the medical care of the future. I have an idea that, whether we like it or not, it's going to be "just us chickens" who will fund the system. It's also clear that we will not have the resources available to provide care to all of us at the present level. The present trend toward outcomes re-

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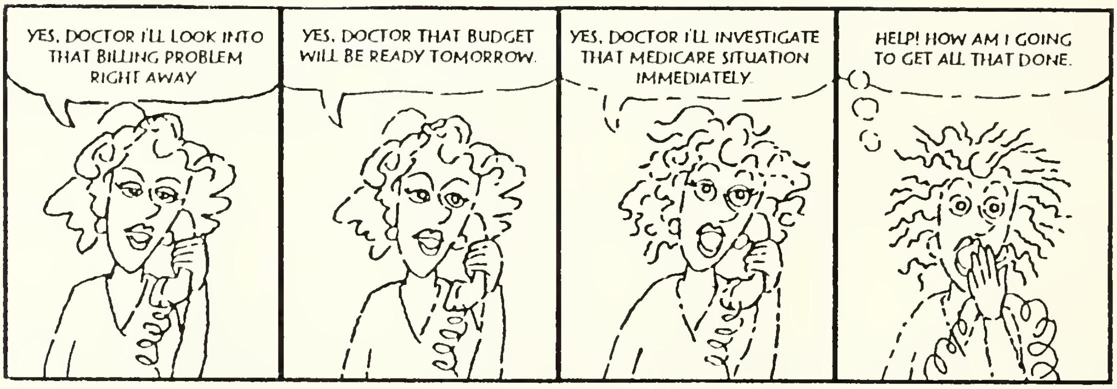
search is the first logical move toward identifying treatments and procedures which should be funded - providing a scientific basis for rationing.

Will we be all right? The real answer to that question requires that we act much like that miraculous little organism, the Missouri crawdad. For that little "animal" has the ability to look backwards as it goes forwards. If we look backwards and remember ultimately why we wanted to be physicians in the first place, the fact that it did happen grants us a sense of permanent power - we actually did it. We wondered how it would be when our lives turned out. Well, they already have. They've turned out just the way they are, right now. The profession is a noble one, as it has always been. We represent it and ourselves every day in our increasing abilities to empower and facilitate people in the domain of their health and well-being. And there is much room for improvement. What a fabulous opportunity - to take on handling the health care of an additional twenty to thirty million patients and to do it in a financially responsible way.

Will we be all right? Not to worry!

Editor's note: Heraclitus in 500 B.C. noted that "nothing is permanent but change". With this in mind the editorial baton will pass this month to the able hands of J.Regan Thomas, M.D.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THE PRESIDENT'S PAGE

by
Dorothy D. Reister, M.D.
Missouri State Medical
Association President

We must be involved in the decisions that will determine Medicine's role in society

A bit of 1992 is behind us and many changes already have occurred. Our "alphabet soup" has been stirred to put into effect significant changes; many of these changes seem destined to confuse us. The reimbursement rate from Medicare was predicted, but no one will know exactly what one's reimbursement will be until HICFA sends a check. Second, there is tremendous revision of coding. This has necessitated numerous courses offered by hospitals, medical societies, HICFA and many private offerings. Certainly it seems we should get CME credit for these! The new changes were to be in place by January 31, 1992 but hopefully we will be given a little longer grace period. Then there is the RBRVS. This document was the result of years of effort by many, many physicians. After spending this time developing, refining, reworking and submitting this document, for many of those physicians, it bears little resemblance to their goal. Again, medicine was over-ridden by government.

Fourth, the budget and expenditures of the state of Missouri have been cut. The state could not find funds to match the federal funds ($300,000) to bring the Missouri coding system into the Federal one. So Missouri claims are submitted with an antiquated coding system. The Health Department cuts included subsidies to local health departments of 16%, testing of public water supplies was DISCONTINUED, three dozen workers were laid off, including some nurses. Mental Health had to cut $38 million, had to shut down the Nevada State Hospital, and almost 200 jobs remain unfilled in other facilities.

Fifth, we have the Missouri State Board of Healing Arts with its regulations not always understood, its investigators suddenly appearing in our offices and although we were promised courteous treatment of physicians and office staff, we still have complaints of the lack of courtesy. We still have physicians complaining about the long delay in hearing from the board. We had hoped to have our liaison committee meet with at least two members of the board in January, but it seems it will have to be February.

What I have done is touch just the tip of the iceberg of state and federal interference with our practices and in our patient care. These are some of the reasons for being actively involved with our Senators and Representatives. We can be Key Contacts; we can correspond frequently; we can inform legislators of the true facts and ramifications of their proposed legislation. We can support our PAC, become sustaining or even super PAC members. The more we stay in contact, the greater our influence. We can even ask how Senator Bob Dole was hospitalized nine days for prostate surgery? And how did Congressmen get a $4,000 raise so quietly? We can all visit our legislators and congressmen at their offices when they are home.

So, hop on our medical-political bandwagon and help! ■

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10220 N. Executive Hills Blvd., #442
Kansas City, MO 64153
816-891-6602

AUXILIARY MESSAGE

by Elizabeth Huffaker
MSMA Auxiliary
Health Projects
Chairman

Family Violence

When we open the newspaper in the morning, we are confronted with news on murder, rape, drugs, or some form of violence. We can not help but be aware that violence is becoming a way of life and a definite societal problem. Yet most are not really aware of the toll it takes on the lives of those who are involved as well as on the community.

Family violence is very complex and as life-threatening as any of the killer diseases of the past two centuries. Even more so than aids, violence could be called the disease of the '90s. What is so frightening is how much of it takes place between people who know each other . . . friends, husbands and wives, parents and children. It has been reported that the American family and the American home are perhaps as/or more violent than any other single American institution or setting (with the exception of the military . . . in time of war.)

According to the FBI:

. . . A woman is beaten every 12 seconds in the U.S.

. . . Over 4,000 women are killed each year at the hand of their batterers. (Battering is the most underreported crime in the country.)

. . . Battered women are more likely to suffer from depression, anxiety, drug/alcohol abuse, psychiatric problems. Attempted suicide, in particular multiple attempts, are significantly higher among abused women.

Statistics show that domestic violence touches as many as one-fourth of all American families. Six of ten couples experience violence at some time during their marriage, with either husbands beating wives, or vice versa. Approximately 900,000 parents are beaten or abused by their children each year. Child homicide is now among the five leading causes of death in childhood, with the majority of infant victims killed by parents, relatives, or older children. These statistics from the AMA Auxiliary on Family Violence are reported here to point out the prevalence of violence in our society and to show that this is a pressing health issue that must be addressed.

Annual estimates from reported domestic violence injuries (taken from the *American Journal of Public Health*, 1989) include:

. . . 21,000 hospitalizations-99,800 days of hospitalization

. . . 28,700 emergency room visits

. . . 39,000 physician visits

. . . $44,393,700 total annual medical costs

. . . 175,500 days lost from work

Notwithstanding the physical and emotional trauma of domestic violence we can see that there is quite a monetary drain on the health care system. With the shrinking health care dollar, monies are being taken from a system that could be spend elsewhere to combat disease.

Realizing the magnitude of family violence in our country and the need for the medical community to become involved in providing help for the victims of abuse, the AMA is asking physicians to become members of the National Coalition of Physicians Against Violence. They will be working with other concerned groups to develop support groups and prevention programs.

The AMA Auxiliary, in response to the AMA's request to join with them to help combat violence in our society, has launched a three-part program that includes educating the public, supporting victims, and providing physicians with resources for

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Auxiliary Message

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their patients who are victims of family violence. Auxiliary members will be invited to join the National Coalition of Physicians Against Violence and to work with physicians to develop local Family Violence Prevention Committees.

The Missouri State Medical Association Auxiliary is very concerned about the violence in our state. Auxilians in different counties are manning crisis hot lines and working in battered women's clinics. We have joined in coalition with the Missouri Chapter national committee for Prevention of Child Abuse and 16 other sponsoring organizations to launch a statewide public education program to prevent shaken baby syndrome injuries to infants and young children.

The purpose of this project is to prevent child deaths and serious injuries. We know that shaken baby syndrome is known to be one of the most serious, yet preventable conditions of child abuse. Children who are vigorously shaken by the extremities or shoulders are being seen by physicians with whip-lash induced intracranial and intraocular bleeding. Other injuries resulting from shaking, slamming, or throwing children include learning disabilities, hearing loss, visual impairment, seizures, cerebral palsy, spinal cord injury, brain damage, and even death. Making the public aware of the problem and educating them to the consequences of their actions is mandatory . . . and WE ALL MUST HELP! Written material addressing this issue has been developed and printed and is free of charge.

Physicians, we are asking that you place these free pamphlets in your office so patients can pick them up and read them. They briefly explain to parents and child care-givers, the dangers of shaking children and provide alternative actions to take, other than shaking their children, when they are frustrated and angry. Attractive, glossy, 8½ by 11 inch posters are available to hang in offices, hospitals, etc. A fact sheet has been developed and further explains this syndrome and what we can do.

The solution is PREVENTION. Your help in this state-wide medical education program is needed.

Spread the word:
Prevent Injury . . .
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You may order your free material by contacting your county medical auxiliary or by mailing the following form to Mrs. Elizabeth Huffaker, 134 Pinehurst Estates Dr., St. Louis, Missouri 63141 (1-314-576-3878).

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PRACTICE MANAGEMENT

by Janice K. Woerth, Ph.D.

Do you have a question for Dr. Woerth? Include your questions along with your name, address and phone number and send them to:

Janice K. Woerth, Ph.D.
Practice management
c/o *Missouri Medicine*
P.O. Box 1028
Jefferson City, MO 65102
Or call her direct:
202/363-1780

Although your name will not be used in *Missouri Medicine,* please include your name and phone number so that Dr. Woerth may contact you if she needs additional information to answer your question.

Dear Dr. Woerth,

We have heard that Medicare is reimbursing for surgical procedures under a "global package". Could you please explain what this means? Also can you enlighten us as to how to use the new modifiers that are in the 1992 CPT book?

D.G. and C.W.

Dear D.G. and C.W.,

Starting January 1, 1992, Medicare began reimbursing claims based on new Global Surgery guidelines. Under these guidelines, most surgical procedures are reimbursed as one "package". This is not a new method of reimbursement for Medicare. However, definition for global surgery payment has been slightly expanded and is important to understand to avoid "unbundling" of charges. A global surgery payment includes the following:

All preoperative visits related to the surgery that are done by the surgeon, in or out of the hospital, one day prior to the surgery;

All intra-operative procedures;

Treatment of all complications not requiring additional operating room time;

All postoperative visits done in or out of the hospital within 90 days following the surgery, including ICU care.

Consultations are not part of a global surgery package and can be billed separately.

The following new modifiers were developed for use with surgical procedures falling under this "package" concept.

-24 *Unrelated evaluation and management service.* This modifier is used when the E/M services rendered in the postoperative period were unrelated to the original surgical procedure performed. An example would be a hospital visit for cardiac dysrhythmias following gall bladder surgery.

-25 *Significant, separate identifiable evaluation and management service by the same physician on the day of a procedure.* Use this modifier when the patient's condition requires E/M services beyond the usual preoperative or post operative services associated with the procedure rendered. An example would be afternoon office visit for chest pain following a morning office visit for an annual pelvic and pap smear.

-78 *Return to the operating room for a related procedure during the postoperative period.* When another surgical procedure is performed within the 90 day postoperative period of the initial surgery that is related to the initial surgery, this modifier should be added to the second surgical procedure. An example would be surgery for postoperative hemorrhaging following coronary artery bypass.

-79 *Unrelated procedure or service by the same physician during the postoperative period.* When an unrelated procedure or service is rendered within a 90 day postoperative period, add modifier -79 to the code of the unrelated procedure. An example would be arthrocentesis for shoulder following arthroscopy of the knee.

The following CPT modifier was developed by the AMA for use with the new 1992 evaluation and management (E/M) codes:

-21 *Prolonged evaluation and management services.* This modifier is only used with the highest level (Level 5) evaluation and management services. When added to a level 5 service, it indicates that more than a level 5 E/M service was rendered or that the service was prolonged.

There are three other modifiers that have been developed for use with surgery codes that have existed for some time yet, these are not often used. These should be used when two or more physicians are involved in surgical care of a patient. They are as follows:

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Practice Management

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-54 Surgical care only. This modifier is added to the surgery code when the surgeon is only performing the surgery and another physician is performing the preoperative and postoperative care.

-55 Postoperative management only. When one physician renders only the postoperative care, this modifier is added to all postoperative services.

-56 Preoperative management only. When one physician renders the preoperative management only, modifier -56 is added to the preoperative services.

Proper use of modifiers communicates to the insurance carrier any deviation or modification the physician has made to a service or procedure. This information can be helpful in efficient and timely processing of Medicare claim forms.

MSMA Patient Brochure Information

MSMA has available several patient information brochures. Topics range from breast cancer treatment alternatives to health care cost containment. You may preview sample copies free of charge before ordering. Submit orders and sample requests, by title, on your professional letterhead, to MSMA Brochures, P.O. Box 1028, Jefferson City, MO 65102. Or, you may fax your orders to the MSMA office at 314/636-8552.
Here are just a few of the titles:

A WOMAN'S CHOICE. Presents various breast cancer treatment alternatives in clear layman's language, discussing advantages and disadvantages of each treatment choice. *Cost: 15 cents each.*

HEALTH CARE COST CONTAINMENT. Do your patients understand why health care costs are rising? Do they realize they can do something about it? In simple language, this popular brochure explains some of the reasons for spiraling costs and shows how the patient and physician working together can help contain them. *Cost: 10 cents each.*

PATIENT SURVEY. These small brochures are designed to elicit your patients' honest opinion about your practice. The answers to the quick survey can provide you and your staff with valuable feedback regarding the operation of your practice. Consider enclosing them with patient bills. *Cost: 10 cents each.*

U.S. Department of Health and Human Services Issues Final Medicare and Medicaid Anti-Kickback Safe Harbor Regulations

James G. Wiehl, Mark H. Goran
Sonnenschein, Nath & Rosenthal, St. Louis, Missouri

On July 29, 1991, the U.S. Department of Health and Human Services, Office of Inspector General, published in the Federal Register final regulations regarding Medicare and Medicaid anti-kickback safe harbors. The long-awaited regulations, mandated by Congress in 1987, are intended to protect business arrangements from being the subject of civil or criminal prosecution under the Medicare and Medicaid anti-kickback statutes. The language of those statutes broadly prohibits anyone from intentionally paying or receiving anything of value to influence the referral of Medicare or Medicaid business. Because of the breadth of the statutes language, many health care providers have been seeking guidance on ways that they can engage in business arrangements without invoking the scrutiny of the Office of Inspector General under the anti-kickback statutes.

The final regulations create eleven safe harbors in the following areas:

Investment Interests
Space rental
Equipment rental
Personal services and management contracts
Sale of practice
Referral services
Warranties
Discounts
Employer-employee relationships
Group purchasing organizations
Waiver of beneficiary coinsurance and deductibles

The final regulations do not include any process for the issuance of advisory opinions. In addition, the comments in the final regulations remind providers that prior advisory letter may not be regarded in any way as authoritative, and the only authority to legalize conduct is the safe harbor regulations. The comments also suggest that the Office of Inspector General will use its discretion to be fair to providers who have previously structured arrangements which do not comply fully with a particular safe harbor, but who work with due diligence and in good faith to restructure the arrangement so as to comply. This article will focus on the first five of the eleven safe harbors listed above, since these are the areas which are most likely to affect the business dealings of physicians.

The following is a brief summary of key elements in each of those five safe harbors. Please note that the following is intended to provide summary information about this new development in the law and does not constitute specific legal advice.

Investment Interests. The final regulations retain a safe harbor for large publicly traded entities. In order to qualify, the entity has to have at least $40 million in assets (the asset test was increased from the $5 million test under the proposed regulation; however, the requirement of a minimum of 500 investors was eliminated from the final regulation). In addition, the investment must meet the following five standards:

(i)Equity securities must be registered with the Securities and Exchange Commission;

(ii)The investment interests must be obtained on terms equally available to the public through trading on a registered national securities exchange;

(iii)The manner in which the entities services are marketed and provided cannot preferentially favor investors;

(iv)The entity cannot loan funds to, or guarantee a loan for, an investor who is in a position to influence referrals, furnish items or services to or generate business for the entity referral or control position) to finance the investors investment; and

(v)The amount of distributions to an investor must be directly proportional to the capital contributed by that investor.

The final regulations also include an additional investment safe harbor for other entities. There are four key defined terms in connection with this safe harbor:

Active investor means an investor either who is responsible for the day-to-day management of the entity and is a bona fide general partner in a partnership under the Uniform Partnership Act or who agrees in writing to undertake liability for the actions of the entities agents acting within the scope of their agency.

Investment interest means a security issued by an entity, and may include the following classes of investments: shares in a corporation, interests or units or a partnership, bonds, debentures, notes or other debt instruments.

Investor means an individual or entity either who directly holds an investment interest in an entity, or who holds such investment interest indirectly by, including but not limited to, such means as having a family member hold such investment inter-

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Safe Harbor Regulations
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est or holding a legal or beneficial interest in another entity (such as a trust or holding company) that holds such investment interest.

Passive investor means an investor who is not an active investor, such as a limited partner in a partnership under the Uniform Partnership Act, a shareholder in a corporation, or a holder of a debt security.

If an entity has either active or passive investors, then the following eight standards must be met to fall within this safe harbor:

(i)No more than 40% of each class of investment in the entity may be controlled by physicians and others who are in a referral or control position;

(ii)The terms on which the investment interest is offered to a passive investor in a referral or control position (e.g., price and quantity of shares) are no different from those offered to passive investors not in a referral or control position;

(iii)The amount of shares offered or other terms of the investment interest cannot be linked to the volume of referrals or business generated;

(iv)The entity does not impose any requirements on a passive investor to refer or generate business;

(v)The way in which the entity markets and furnishes its services cannot preferentially favor the passive investors over non-investors;

(vi)No more than 40% of the revenue can come from referrals or other business generated by investors;

(vii)The entity cannot loan funds to, or guarantee a loan for, an investor who is in a referral or control position if any part of such loan is used to obtain the investment interest; and

(viii)The amount of the dividends distributed must be directly related to the capital contributed by the investors.

Physicians would be well-advised to evaluate all of their investments to determine if any of them put the physician at risk in light of the safe harbor developments. Of particular concern are any investments in entities with which the physician has business or professional relationships other than the investment itself. Moreover, a physician's investment, in an entity that doesn't come within a safe harbor, can be at risk even if that physician is acting in a manner so as to conform with safe harbor requirements. Because substantial penalties can be imposed on violators, physicians will likely find it advantageous to seek counsel in weighing the risks against the potential return on the investment.

Space Rental, Equipment Rental, Personal Services and Management Contracts. Although three separate safe harbors are delineated under the final regulations, the major elements of each are virtually identical, as follows:

(i)There must be a signed written agreement;

(ii)As to rental of space or equipment, the lease must specify the premises or equipment covered; as to personal services or management, the agreement must specify the services provided;

(iii)If access to the space or equipment, or services to be provided, is on a periodic time basis, then a schedule for those intervals of use or service, the exact length of such intervals, and the exact payments associated therewith must be specified;

(iv)The term for the agreement is for at least one year; and

(v)The total amount of payments are set in advance and are based upon fair market value in an arms-length transaction, without taking into account any referrals.

Under this last standard, the definition of fair market value does not permit the rent or charge to reflect any value attributed by either party to the proximity or convenience of the provider to the practitioner who is in a position to make referrals. The safe harbor for personal services and management contracts includes an additional standard, which prohibits the counseling or promotion of any business arrangement that violates any federal or state law.

All contracts between physicians and other providers, potential referral sources, or vendors should be closely reviewed for safe harbor compliance. Even if no kickback or other improper benefit actually exists, even the appearance of impropriety can lead to serious consequences. Loosely structured or "handshake" deals should be formally documented and restructured, if necessary, to reflect the bona fide nature of the relationship.

Sale of Practice. The final regulations contain a limited safe harbor in connection with the sale of a practitioners practice to another practitioner provided two standards are met:

(i)The sale is completed within one year from the date of the first agreement; and

(ii)After one year from the date of the first agreement, the selling practitioner will no longer be in a professional position to refer or generate business.

The final rule addresses practitioner to practitioner sales, and therefore does not appear to offer any safe harbor to a hospital acquisition of a physician practice. It is important to note that the time periods run from the date of the first agreement, not the closing. For example, if an agreement regarding the purchase and sale of a physician's practice is signed in January, 1992, but not closed until July, 1992, the sale must be completed and the selling physician must no longer be in a position to refer by January, 1993.

Finally, it is important to realize that while the Medicare and Medicaid anti-kickback statutes are very broad, not every transaction necessarily comes within their reach. Thus, failure to come within a safe harbor does not necessarily mean a transaction is illegal. The safe harbor regulations merely provide protection from prosecution for transactions that come within one of the safe harbors. However, the message from the Office of Inspector General ("OIG") is clear that dealings within the safe harbors, or restructured to come within them, will be more favorably looked upon than those transactions or business arrangements that do not. While the OIG has indicated that additional safe harbors may be issued in the future, prudence dictates a thorough review of business arrangements with respect to the safe harbor regulations as they currently exist.

MOMEDICO: An Answer To A Need

Richard V. Bradley, M.D., President and CEO of MOMEDICO

Medical Professional Liability A Missouri History

Prior to 1962, relationships between patient and physician were based upon a contract of mutual understanding. Untoward results were understood to be within the realm of reasonable risk. For potential liability for medical activities below standard, physicians maintained policies for medical professional liability.

Between 1962 and 1973, the legal community was involved in a somewhat inconspicuous process of converting the well-established laws of contract to laws of tort. Written warranties on many products such as lawn mowers, autos, and electrical appliances, became essentially useless when subjected to the vagaries of trial by jury.

Although claims of product liability lead the way, medical professional liability claims increased in *"frequency"*. Jury awards against physicians, and hospitals, increased in *"severity"*.

Commercial insurance companies, providing medical professional liability coverage, faced two problems during the period 1972-1974. (1) Investment portfolios were performing poorly enough to warrant reevaluation of company policy. (2) Future projections of professional medical liability claims activity were not favorable. In fact, indications wee terrible.

It should be noted that some commercial companies had correctly considered medical professional liability insurance as a *"Cash Cow"* for a period of five decades. These companies had established a considerable positive cash reserve from which to handle claims in the area of medical professional liability. In close chronological order, and sequence, the commercial carriers began to unload all, or the major part, of their medical professional liability book of business. For the accounts that were maintained, harsh underwriting standards were imposed together with collateral demands for other portions of the applicants insurance portfolio such as auto, home, and/or health.

Approximately, two-thirds of the doctors in Missouri found themselves looking for medical liability insurance coverage between 1973 and 1975. The worst aspect of this problem was that there was essentially no market available.

For Physicians, who entered practice prior to 1973, this report will serve as a reminder of the problems with which they dealt during the period 1973-1975 in the area of medical professional liability insurance.

For physicians, who entered practice after 1975, this report can serve as historical reference well worth serious consideration.

In 1975, the Missouri Legislature demonstrated an attentive attitude to the unavailability of medical professional liability insurance. The "Spradling Amendment" which resulted, allowed a small group of professionals to form a mutual assessable casualty insurance company which could assess its members retroactively for any or all losses incurred. Another part of this amendment prohibited plaintiffs from demanding a specific dollar amount for alleged damages, (better known as a *"prayer"* in legal terms).

Several groups of physicians and hospitals availed themselves of the ability to form a mutual assessable insurance company. This legislation was assigned a statutory chapter number of 383. Thus the companies became known as "383" companies.

The Missouri State Medical Association, House of Delegates, Staff, and Council were all active in the 1975 legislation due to a considerable amount of hard work by the *Liability Crises Committee for Tort Reform*. This was not an army. It was more of a "SWAT" team made up of members of the many component medical societies making up the MSMA federation.

The MSMA Delegates, Staff, and Council did not wish to form a mutually assessable "383" company. Instead, they elected to form a captive stock company which would be totally owned by the MSMA. the members of the

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MOMEDICO
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MSMA had serious reservations about an insurance company which could legally come back, in the future, and place a monetary call on them, or their estate for past events. Thus, MSMA set about the incorporation of the Missouri Medical Insurance Company.

Formation of The Missouri Medical Insurance company (MOMEDICO) was requested by unanimous resolve of the MSMA House of Delegates. The charge was to incorporate a stock company, wholly owned by the MSMA, which would remain solvent for available insurance by Missouri physicians at a premium which would be appropriate. The MSMA required that each insured physician would purchase a non-interest bearing debenture, subordinated to specific conditions with the MSMA, in the amount of $1,000 for each class number required by that physician's insurance risk. ($1,000 for Class I and $6,000 for Class VI) These debentures became a contract between the insured physician and the MSMA. MSMA secured a loan in the amount of $2,500,000 with its stock in the company, at prime interest plus 1%. The loan specified a formula for the payment of interest and principle. Prime interest rates of 9.5% (1978) accelerated to 19 or 20% within 18 months. Interest and principal payments exceeded the fiscal means of the MSMA. In addition to this untoward economic event, the availability of professional liability insurance, from other sources resulted in allowing the payment of the debentures to be spread over a three-year period, in order to effect their sale. Total debentures sold by MSMA amounted to $2,016,300, which was less than the bank loan mentioned ($2,500,000). Clearly, additional sources of funds were needed in order for MSMA to pay off the loan made from Boatmen's Bank in 1978.

The Board of Directors of MOMEDICO, working with the Council of MSMA, developed a plan for raising additional funds via a stock offering, designed to obtain the support of Missouri doctors:

1)MSMA would relinquish total ownership of the company but;

2)MSMA would maintain voting control of the company through retention of a limited special class "B" stock wherein each share carried 15 votes on company matters while regular class "A" stock had 1 vote per share.

3)Doctors with paid up MSMA subordinated debentures could exchange $20 of debenture for one share of class A MOMEDICO stock.

4)For a holding period of more than 180 days, only Missouri physicians could purchase MOMEDICO Stock (This was published in almost every community paper and broadcast on almost every licensed radio station in Missouri with personal visits by an officer of the company)

5)After the holding period mentioned, stock was available to the general public.

The final result demonstrated that some 98% of MOMEDICO's Class A Stock was purchased by Missouri doctors, MSMA, or physicians' staff and families.

Class B shares of stock were issued to MSMA to represent the original intent of foundation and sponsorship of the company.

MOMEDICO further utilized brokerage fees to MSMI, a subsidiary of the MSMA, in order to provide funds for the payment of interest and principal which MSMA owed to Boatmen's Bank. These monies came from the funds derived from the sale of stock. This was the continuation of a loan process, begun in the late 1978 and 1979 by MOMEDICO, to allow MSMA to make loan and principle payments to Boatmen's Bank.

MOMEDICO paid, to MSMI, brokerage fees of $767,492 which amounted to $238,239 in excess of total debt service incurred by MSMA on the principal and interest owed by MSMA to Boatmen's Bank.

Ultimately, MSMA came to have a 72% ownership of stock based upon par value ownership of stock in MOMEDICO and a 61% voting control of the company.

MOMEDICO Class A stockholders have belonged to two groups between 1980 and 1991. One group continues to hold stock and has supported the company with loyalty to the purpose for which the company was founded. The other group of insureds of the company have divested themselves of stock by retirement, death, disability and, yes, a few departures.

Economic History of MOMEDICO

From its inception, MOMEDICO was dedicated to:

1)Providing for the aggressive defense of physicians against non-meritorious allegations of "Malpractice";

2)Achieving fiscal solvency; and

3)Living up to the charge presented to the company in the beginning. *To maintain a solvent, durable, source of Medical Professional Liability Insurance coverage for doctors [of Missouri] at an appropriate premium.*

These requirements were attended to by experienced insurance professionals selected and supported by the Board of Directors of the company. As of 1980, MSMA continues to serve as the parent organization of MOMEDICO plus a body of 300 to 400 stockholders.

MOMEDICO did not enjoy a profitable year until 1987. For the first 9 years of the Company's existence, its shareholder received no return on their investment. Only after the timely evaluation of this turn of events did MOMEDICO enjoy the opportunity of considering any reward for either insureds or stockholders.

The *first effort* was to reward insured physicians who had been able to practice medicine in a personal environment free of claims alleging medical malpractice. this reward consisted of premium discounts, from base premium, for periods which were free of claims.

The *second effort*, also a reward, was to demonstrate to those doctors insured in the first profitable year, that the participating clause of MOMEDICO's policy was something more than talk. A partial return of premium was distributed to every MOMEDICO insured who had a policy in force for any part of the year 1987 whether they were, or were not, insured at the time the funds were distributed in 1989. This distribution dealt with a special surplus stabilization premium which enabled MOMEDICO to do just that stabilize the monetary surplus which is vital to a successful medical professional liability insurance company.

The *most recent reward-effort* was a special stock dividend voted by the Board of Directors of MOMED HOLDING CO. on May 10, 1991. This was a most nominal return on investment to those loyal stockholders who had demonstrated their loyalty and their patience for some 13 years. The dividends amount to $.25 per share for both class A and class B stock.

The MOMEDICO Board of Directors has remained steadfast in asking its staff of insurance professionals to maintain the solvency of the company through fair underwriting, actuarially sound and appropriate insurance premiums, vigorous defense of physicians against non-meritorious allegations of malpractice, and continued service to insureds through efforts in risk prevention, tort reform, support of physician rehabilitation, and aggressive defense of the high purposes of medicine in the community, Missouri and the United States.

Future intentions:

MOMEDICO is dedicated *first* to aggressive defense of the doctors who are wrongly accused of malpractice. At the same time, it should be acknowledged that equitable compensation and rehabilitation of those patients whose claims have merit is agreed upon by both the company and the insureds.

Second: MOMEDICO is dedicated to permanence of available medical professional insurance so long as potential insureds desire the same, are willing to improve the practice of patient service and, want our company to work with them at a premium level which is actuarially adequate.

Third: MOMEDICO is dedicated to risk prevention through orientation, education and self assessment. This must work hand in hand with active efforts toward legislative tort reform. In 1985-1986, a coalition effort by organized medicine, organized hospitals and organized doctor-controlled insurers did achieve helpful tort reform through compromise. Objective analysis provides proof that the efforts were beneficial. there is more to do!

Fourth: MOMEDICO is dedicated to reward insured doctors who serve their patients in a personal environment free of avoidable misunderstanding with resultant freedom from claims of malpractice. This is achieved through reduction of base premiums for classes as well as through claims-free discounts for individuals.

Fifth: MOMEDICO is dedicated to cost-effectiveness in management and administration. Careful scrutiny is given to the overall usefulness of any project in relation to the goals of the company.

Sixth: In 1991, MOMEDICO was able to arrange the (staggered) method of election of members of our Board of Directors in a way which will allow that Board to continue active participation by representatives of Missouri State Medical Association Council who have been, and will be representatives of the component medical societies of Missouri.

Seventh: The stockholders of the MOMED HOLDING COMPANY today represent the stockholders of MOMEDICO. The stock was exchanged share for share at no cost to any stockholder as a method of making our economic base of operation more effective. In the future, the 95% ownership of MOMED HOLDING CO., currently made up of the MSMA physicians, MOMED itself, and close relations or staff may well become diluted due to purchase by entities outside medicine. Why, because MOMED HOLDING CO. is truly a growth stock. Please recall two important facts about stockholder control of the company. It is controlled by MEDICINE. MEDICINE can retain that control. MEDICINE can thus continue the election of the Board of Directors. For all future directors may the present Board offer its record as a challenge which, if met and improved upon, will truly be appreciated.

Information about MOMEDICO has always been available for those who are truly interested and willing to ask. Unanswered questions may be those which were, in fact, never asked.

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IS THE PRO A QUACK?

John Layle, M.D.

Many of us hoped when the PRO was formed, in 1982, that we would now have professionalism with teeth. We believed, or at least hoped, that docs would now be able to police themselves according to science-based professional standards armed with the enforcing power of a governmental regulatory agency. We hoped we would finally be able to rid our ranks of the relatively small number of quacks, incompetents and exploiters whose misbehavior tarnishes us all. We hoped the PRO would allow us to defend our profession against charges of unnecessary care, waste and poor quality. We believed the new program would be fair because docs would be policing docs, and that it would be effective because the decisions of the PRO would ben enforced by the regulatory and punitive power of the U.S. government. Alas, the evidence is now overwhelming that in many states, Missouri among them, the PRO itself has taken on some of the characteristics of quackery which it was intended to abolish and the net results of the program despite its good intentions are lower quality and higher cost than we would have had if the program had never been started. Indeed, the number of charges against the PRO and the growing volume of evidence supporting those charges is now so large that it cannot be adequately summarized in a single essay such as this but we must try somehow to push the debate about this new administrative debacle into the forefront of the public agenda before the very idea of professionalism drowns in its own cynicism.

Somehow the public must be made aware of the fact that the PRO is evolving into the same kind of regulatory swamp we have seen all too often when groups of people who, as individuals may be well-meaning, law-abiding, respected and educated, nonetheless become corrupted by a kind of myopic mob psychology that focuses on the regulatory trees and ignores the social forest. This corruption results from the assignment of broad, ill-defined powers to investigate and punish outside, or in parallel with, the ordinary courts and police system. It occurs when a system of investigation and punishment is established not so much to protect the rights of individuals from infringement as to impose the ideologic, political or managerial will of the state upon the behavior of individuals. It is not a phenomenon confined to monarchies, theocracies and despotisms but is a part of many bureaucratically dominated democracies. It occurs in democracies when laws are made, interpreted and enforced by non-elected officials but instead of laws they are called "regulations". It is, in short, a bureaucratic quasi-justice system that exists alongside or instead of a system of justice. It does not require any rules of evidence, trial by jury, presumption of innocence or binding by precedent, and it does not place the burden of proof upon the prosecution. It is rationalized by the principle that the desirable ends justify the deplorable means. Paradoxically the deplorable means ultimately prevent the desirable ends. Thus, it creates the quintessential social paradox: it is the road to hell paved with good intentions; inefficiency in the name of efficiency, waste in the name of economy, rationing in the face of abundance; persecution in the name of distributional justice; harassment in the name of order; irrationality in the name of protocol. It is the antithesis of professionalism and it has now, in the case of the PRO, co-opted our profession; it has replaced ethics and morals with regulations and legalisms. It has replaced the goals of the profession with the goals of the government bureaucracy.

After seeing how the PRO works it is no wonder that the *administrative* costs of our ponderous health care system increased a whopping 26% last year, over twice as fast as overall medical costs. It is no wonder that the Commerce Department projects that *administrative* costs will continue to increase 20% each year for the next five years. It is no wonder that health costs are much higher since the imposition of the PRO system than they were before. It is no wonder that health costs in Canada, where docs are given much more autonomy, are lower than they are here where docs are so tightly regulated. After seeing how the PRO works we are not surprised to learn that the major reason why health costs are lower in Canada is not because medical care is cheaper but because administrative costs of the payment system are lower. It is no wonder that although docs in Canada make less money and dispense more medical care per doc, they are happier than are U.S. docs. After seeing how the PRO works it's no wonder that applications to U.S. medical schools have been falling for the past decade and that there is a growing shortage of internists and of other primary care docs, a shortage so large that it has been called a "crisis".

When one sees how incompetently the PRO operates it is no wonder that its decisions are nearly always overturned when they are ultimately brought before an independent civil tribunal. The evidence, for anyone who cares to examine the paper mountains of it, leads to only one conclusion: The PRO is simply unable to consistently discriminate between good and bad care. It is unable to discriminate between right and wrong, fair and unfair, logic and illogic, sense and nonsense, science and bias, morality and persecution, the public interest and bureaucratic self-interest. The PRO is an expensive sham that claims, and charges the public for, medical expertise it does not possess. It is, in short, a quack that sincerely believes despite all the evidence to the contrary, its painful manipulations are doing something wonderful.

Over the past few years we have received numerous letters and verbal communications from our members describing the abusive, inane and obtrusive way the PRO in Missouri works. Space does not permit an adequate analysis of these letters here, and of course we have no way of checking the facts about individual cases, but all of our personal experience and all of the evidence we have accumulated seems to corroborate them. Indeed the PRO keeps the essential data secret or elects not to discover data which could be used against it. It does not feel compelled to answer our questions, to give its reasons or to defend its reasoning. It does not impose upon itself the constraints of scientific logic based upon observable evidence but makes up its own rules, often out of thin air, and applies them in a supercilious and capricious way. When confronted with evidence of incompetence the local units blame HCFA and HCFA blames local units. The staff blames the docs and the docs blame the staff. All hide behind rules they individually disavow. The docs say "If we don't do this terrible thing, someone else will only do it worse". PRO authorities do not dispute the fact that the PRO is incompetent they simply claim that if they tinker with the system some more they will eventually get it right:

"Presently we are working under a very antiquated review system," said Thomas Morford, director of HCFA's Health Standards and Quality Bureau. "Our tools are limited to the judgment and experience of nurses and physicians conducting review. Because this judgment varies, so does the caliber of review". (Am. Med. News, Nov 9, 1990)

Yet, if you talk to those very nurses and doctors who conduct the reviews they say they are forced to do stupid things because they are responding to inane directives from incompetent superiors in the mindless HCFA hierarchy. thus HCFA has superimposed a pervasive institutional incompetence as a remedy for the individual incompetence of a few doctors. not surprisingly the very people who created and control the current administrative train wreck endlessly propose to make things better by coming up with a "new system". Andrew Weber, executive vice present of the American medical Peer Review said,

"The hope and promise of this [new] system is that it will move us beyond individual case findings."

The salient feature of their belated acknowledgement that they have exercised misjudgment and unwisdom on a massive national scale is that the people responsible for the mess under which we currently suffer do not have the grace or moral courage to resign. Of course in a true democracy incompetent leaders can be voted out of office; in a free market the people can choose to patronize other establishments; in a free country one can move away from oppressive local government or can enlist the protection of the federal justice system; with a free, unbiased press the incompetence of office holders can be exposed to public ridicule and they can be hounded out of office; but under a monopolistic, autocratic, centrally controlled bureaucratic hierarchy such as HCFA's there are no free elections of top leaders; one can't move away from its jurisdiction; one cannot choose another vender; our news media, which is, in effect, a fawning tool of the health bureaucracy, simply doesn't understand. Thus we are left with this: it is up to us to exercise the responsibilities of a profession. It is up to us to elucidate the truth among ourselves using the modest tools available even though no one else will listen. It is up to us to publicly describe what we see and invite open scrutiny, criticism and analysis of our observations and conclusions. We must believe that ultimately truth will out.

A CARICATURE EXAMPLE

Here's how, in caricature, the PRO works: Suppose that a man crippled by childhood polio were discharged from the hospital after being treated for say, pneumonia. The day after discharge he is struck by a truck and readmitted. The PRO might say to the doc, "You discharged that patient prematurely" implying, without actually saying so, that if the patient had remained in the hospital he wouldn't have been injured. The PRO would point out that the patient was still crippled at the time of discharge implying that he couldn't get out of the way because he was crippled, whereas if he had remained in the hospital, presumably until his agility returned to normal, he might have gotten out of the way. The PRO would, on the basis of this nonsensical *post-hoc-ergo-propter-hoc* reasoning, threaten to withhold payment for the second hospitalization. (He had the disease during the first hospitalization; the disease caused the second hospitalization; therefore the second hospitalization shouldn't be paid for). Yet in the very next case, another man, also with post-polio crippling, also discharged after treatment for pneumonia, also struck by a truck and also readmitted, the PRO might come out *with the opposite conclusion*. It is in other words an irrational system which explicitly allows arbitrary and capricious decisions against doctors, hospitals and patients and yet amazingly is sponsored and supported by organized medicine.

A REAL EXAMPLE AND A CHALLENGE

To give a first-hand real example of how the PRO works in Missouri what follows is, verbatim, a defense I recently submitted in response to an allegation by the PRO that I prematurely discharged one of my patients. I certify herewith that all the facts contained in this letter are true and that I have not knowingly omitted any essential details. I invite the PRO to publicly confirm, or refute point by point, the facts and/or conclusions stated below.

CASE REPORT

To: Ralph O. Smith, M.D.
Missouri Patient Care Rev. Found.
505 Hobbs Rd., Suite 100

Jefferson City, MO 65109
November 12, 1990

Re: Patient XXXXXXXX
No.: Case # XXXXXXXX

Dear Dr. Smith:

You wrote me alleging that my patient was prematurely discharged because she had a positive urine culture untreated, elevated platelet count, low serum sodium, low albumin and low total protein and was discharged without an indication that oxygen was being continued. You state, "before we make our decision we would like to give you the opportunity to submit additional pertinent comments". They are these:

Summary of the case.
The patient's chart contains the following information:

This lady is 89 years old. She is crippled from longstanding rheumatoid arthritis and suffers from severe osteoporosis with multiple compression fractures, kyphosis, bronchiectasis, COPD, stress incontinence, oral thrush (presumably secondary to her multiple previous courses of antibiotic given for pulmonary and other infections prior to admission) and intermittent diarrhea (presumably from the same cause). She was admitted to the hospital because of a broken hip and had a femoral head replacement by the orthopedic service. Her length of stay in the hospital was 14 days. In addition to the orthopedic service and rheumatology service she was seen in consultation by pulmonary specialists. Less than 24 hours before discharge, at 6 p.m., a nurse reported to the orthopedic resident that the patient was incontinent of urine. He ordered a urinalysis and culture on a catheter-obtained specimen the results of which were not available until after she had gone home.

Yes, the urine culture was positive (E. coli 100,000 colonies).

Yes, that positive urine culture was "untreated" during that hospitalization.

Yes, her platelet count was moderately elevated (657,000).

Yes, her serum sodium was mildly low (131 mEq/1).

Yes, her serum albumin was moderately low (2.8 gm/dl) as was her total protein (5.9 gm/dl).

Yes, she was discharged without indication that oxygen was being continued at discharge.

Comments.
1. Regarding the urine culture: while I readily admit this patient was discharged before the results of the urine culture were known, I question whether that is a valid reason to allege that she was discharged *prematurely*. Is it really prudent or responsible to keep a patient in the hospital until the results of a pending urine culture are back? You imply that it is; I respond that it is not. How can I defend myself? All I can say is that common sense is on my side. You, on the other hand, are charged, as you admit, by federal mandate to assure that services provided Medicare beneficiaries are "rendered economically, are of a quality that meets professionally recognized standards, and are supported by *evidence of medical necessity*", if you claim I should have kept her in the hospital until the results of the urine culture were available, it is therefore *your* obligation to show *evidence* of medical necessity. I submit that you do not have such evidence.

It is *your* obligation to assure that prolonging this patient's stay, simply to await the results of this test, would be a service *rendered economically*. I submit that you cannot justify prolonging this patient's hospitalization on economic grounds.

You are obliged to assure that such a prolongation of the patient's hospitalization would meet *professionally recognized standards*. I submit that such a prolongation of the patient's hospitalization would not meet any professionally recognized standards.

But leaving all that aside, suppose we had left her in the hospital until the results of the culture were known, would we then be medically obligated to treat her? Remember that her culture was done not because of clear-cut symptoms of urinary tract infection such as dysuria, fever of CVA pain, but because of incontinence, a chronic mechanical problem present for many years prior to admission. Remember that because of her bronchiectasis and COPD she has had, over the years, *many* courses of antibiotics which did not cure the incontinence. Remember also that she was already suffering from problems resulting from repeated previous antibiotic use, namely oral moniliasis and diarrhea. Remember also that the specimen was obtained under less than ideal sterile circumstances: in the evening, in a deformed elderly patient with severe rheumatoid arthritis, with chronic incontinence, poor personal hygiene, who has just had surgery for a hip fracture. She had oral moniliasis and possibly vaginal moniliasis as well. Imagine for a minute the technical problems of the nurse trying to obtain a sterile cath specimen without vaginal contamination. In the absence of clear-cut symptoms of urinary tract infection, is it not possible that the catheter was contaminated with vaginal secretions? Even if it were not, are we really obligated to treat every case of bacilluria? I remind you that Cecil's Textbook of Medicine (which is, I suppose, the most generally accepted expression of current "professional standards") says this about bacteriuria (18th edition, Volume 1, page 630, authors Wyngaarden and Smith, published by Sanders):

"Asymptomatic bacteriuria should probably not be treated except in those patients who are at high risk of developing symptomatic infections. Thus, treatment of asymptomatic bacteriuria is indicated in pregnant patients to prevent symptomatic illness in the third trimester; in patients who may have major predisposing factors to renal disease, such as diabetic or polycystic kidneys, or who have anatomic or neurologic abnormalities; and in patients who are immunocompromised or who will undergo urologic manipulation. If the treatment fails to eradicate asymptomatic infections in such individuals, further treatment should be reserve for acute symptomatic episodes. In contrast, asymptomatic bacteriuria in females should not be treated in the absence of underlying structural or neurologic lesions, since the likelihood that renal damage will occur is slight. Furthermore, short courses of therapy, when effective, are com-

monly followed by reinfection. In addition, asymptomatic bacteriuria in patients with indwelling catheter nd in the very elderly or non-ambulatory patients should not be treated, because the toxicity and expense of therapy may outweigh the risk of disease."

What could be more clear and unambiguous? Perhaps you thought this 80-year-old patient might be pregnant? I will admit we did not do a pregnancy test. I frankly did not ask her about morning sickness or whether she had skipped a period or about her recent sexual history. I submit that, in spite of these omissions, if this 80 pound lady were in her third trimester even a cursory physical examination would have revealed that fact.

I confess further that we did not ask her about polycystic kidney disease, nor did we do radiographic or radioisotope studies to visualize her kidneys to "document" that her kidneys were free of polycystic disease. Perhaps you thought the patient was immunocompromised or that she would undergo urologic manipulation. But I submit there is no evidence to support any of these indications for treating her bacilluria. I submit further that your suggestion that this patient's bacilluria should have been treated ignores the fact that she has relatively strong *contraindications* to treatment and that any reasonable assessment of the risks and costs versus the benefit would dictate that this patient's bacilluria should *not* have been treated. Of course, I could be wrong. I am entirely open-minded and always anxious to learn better and more efficient ways to treat my patients. I would be very glad to see the documentation supporting your allegation that this test result should be treated.

But if you do not have hard evidence that our care in this regard was a substantial deviation from "professionally recognized standards", then I, herewith, accuse you and your organization of bureaucratic charlatanism, I accuse you of violating the very charge under which you operate. I accuse you of creating and encouraging the very waste, inefficiency, poor quality and unprofessional practice which you and the PRO are supposed to prevent, and I accuse you of using your government office to wrongfully invade my privacy, harass and abuse me to no legitimate purpose.

2. Regarding this patient's elevated platelets: Yes, this patient had an elevated platelet count. Perhaps you are not aware that platelets are commonly elevated in patients with rheumatoid arthritis or that rheumatoid arthritis is an incurable disease or that patients with rheumatoid arthritis often carry elevated platelet counts for years or even decades at a time.

Perhaps you are unaware that platelets commonly act as acute phase reactants and increase as a result of a variety of chronic inflammatory conditions such as bronchiectasis or tissue damage such as bone fractures.

Is this patient's elevated platelet count really evidence that her discharge wa premature? Are we really obligated to keep every rheumatoid patient with elevated platelets in the hospital until those platelets fall to normal? What do you presume we should have done to bring her platelet count down? Give busulfan? Total body radiation? And if we could not get her platelet count down short of such drastic measures, how long should we keep her in the hospital, another week? A month? Six? A year? Again I ask you what "professionally recognized standards" have we violated by discharging this patient with an elevated platelet count? If medical services must be "supported by evidence of medical necessity", what evidence would justify keeping her in the hospital longer for an elevated platelet count? If you have such evidence I would be pleased to see it, but if you do not, then I hold that your allegation is arbitrary and capricious, that you and the PRO have behaved in a disgraceful and irresponsible manner. I hold that you owe me an apology and a public retraction. I hold that in fairness and in candor, you should publicly acknowledge that the PRO is unable, as this case abundantly demonstrates, to discriminate between good medicine and bad medicine; that the PRO has, in many cases put the quacks in control of the doctors.

3. Regarding the patient's sodium: As you know (or should know) a patient's sodium may be low because of too little sodium or too much water. in her case there is no reason to suspect sodium deficit since she was not taking diuretics nor did he have cause for sodium wastage. The only reasonable explanation therefore is that she had mild water retention. Water retention in the presence of normal kidneys (her creatinine was 0.6) is either due to too much in, or too little out. In her case there is no reason to suspect excessive intake, thus the only possible explanation is that she put too little out. Water retention, as you know (or should know), is a usual and normal response to stress such as surgery and is a normal response to decreased circulating blood volume as occurs in patients with a low serum albumin such as our patient. Thus there is every reason to believe that her mildly decreased serum sodium is not pathologic but is instead an expected component of her total clinical picture. I remind you that her serum sodium prior to discharge was 131. Is this level harmful to the patient? Let me tell you what another standard textbook of medicine (*Scientific American Textbook of Medicine*, Chapter 10, Section I, Disorders of Waster, Sodium and Potassium, page 7) says about that:

"Most patients with water retention do not have symptoms attributable to disorder. Symptoms of water intoxication can appear, however, when water retention is sufficient to decrease the serum sodium to a level less than 120 meq/L; at levels less than 110 meq/L such symptoms are common. A serum sodium level as low as 84 meq/L has been reported."

Thus this patient's low sodium was neither unexpected nor dangerous. Why then do you allege that she was discharged prematurely? if we had left her in the hospital longer what should we have done to improve the serum sodium? Lots of tests? Should we have started fluid and electrolyte balance? Consulted a nephrologist? Ordered serum ADH levels, intake and output studies, daily weights, serum and urine osmolalities, 24-hour urine collections, clearance studies? Or should we have treated the sodium value without more tests? If so, what would you have us do? Administer salt tablets or hypertonic intravenous saline? Should we have withheld water? Again, I quote from *Scientific American's Textbook of Medicine:*

It appears to me that the serum sodium concentrations need not be raised rapidly if hyponatremia has been present for several days and the patient's central nervous system status is satisfactory. In such a situation, solute loss from the brain has probably prevented significant elevation of the brain's water content. Rapid elevation of the serum sodium concentration will acutely remove water from the brain and thus may cause excessive brain shrinkage; a similar state is observed when hypernatremia develops suddenly in an hyponatremic person. Water restriction alone, aiming for a serum sodium level in the normal or mild hyponatremic range (about 130 meq/L) within one week or so is the most conservative therapy."

Thus, if we had decided to treat this patient with water restriction she would already be at a level, 131 meq/L, which is even better than that recommended as a target, 130 meq/L, by recognized medical authority. How could we possibly have improved upon that?

4. Regarding the hypoalbuminemia: Perhaps you are unaware that in rheumatoid arthritis *"The albumin peak (on the serum protein electrophoresis) is frequently low"*. That information is readily available to you in another standard medical textbook, *Arthritis and Allied Conditions* by Daniel J. McCarty, 10th edition, Lee and Fabiger, page 648. If you bother to check this reference you will learn that not only is low serum albumin a frequent component of rheumatoid arthritis, but that it results from peripheral consumption of albumin and is not a result of inadequate protein intake or decreased production. In fact, the rate of production of albumin in patients with rheumatoid is generally above normal. Her hypoalbuminemia is thus not evidence of undernutrition, it is simply an expected manifestation of her underlying disease. Perhaps you are unaware that rheumatoid arthritis is incurable? In any case, I fail to understand why you claim that her hypoalbuminemia is evidence that she was discharged *prematurely*. If you are proposing that she remain in the hospital until her serum albumin becomes normal, I suggest that she could look forward to a remarkably long hospitalization. In reviewing our office records, I note that her serum albumin was essentially the same (2.9 gm/dl) *as far back as 1980*! What should I have done about her low serum albumin, started parenteral hyperalimentation? Put down a nasogastric tube and force fed her? If you are aware of an effective treatment for this manifestation you should publish it since, as near as I can tell, the scientific medical community has not yet learned of your remarkable remedy.

5. Regarding her low serum protein. Perhaps you are unaware that the serum albumin is the major component of serum proteins. In her case her serum protein is low *because* her serum albumin is low. It is thus redundant to list *both* as evidence that she was discharged prematurely.

6. In regard to the fact that she was discharged "without indication that oxygen was being continued at discharge". It is, of course, unfortunate that someone forgot to document this detail in her medical record. I remind you, however, that this patient did not require oxygen prior to her hip fracture and although she had an episode of confusion postoperatively while she was on a number of medications (Demerol, Darvocet, Halcion, Phenergan and H2 blockers), her mind eventually cleared after these drugs were stopped and remained clear prior to discharge even in physical therapy when she was off oxygen, and walking in a walker. Although she had some hypoxia postoperatively and was started on oxygen it did not cause her confusion to immediately clear. However, as her normal recovery progressed, as she increased her physical activity, after she stopped all sedatives and respiratory depressants, then her confusion cleared. The fact is that all of the people involved in her care (the orthopedic surgeons, rheumatologists, nurses, physical therapists and respiratory therapists) thought she was functioning in a satisfactory way off oxygen at the time of discharge. The respiratory therapist's final note explicitly stated that she could be discharged. I admit that we failed to "document" what seemed to be everyone's clinical impression, that her pulmonary status had been returned to its pre-hospitalization level. Nonetheless there was every reason to believe that the precipitating factors had been removed, that her functional status had been returned to its previous level according to other parameters and there was no evidence whatsoever to the contrary. It is important to emphasize here that she was not being discharged to her own recognizance but to a structured program of daily professional supervision. For all these reasons I do not think we would have been justified to have ordered an additional set of pulmonary function studies, pulmonary consultations or x-rays on the basis of the evidence available at the time to "document", for governmental "second guessers" like you, that she was ready for discharge.

Conclusion

In summary, I challenge your assertion that these tests and treatments, or lack thereof, taken as a group or individually constitute any legitimate basis for your charge that I discharged my patient prematurely. I hold that the irresponsible allegation on your part demeans me and imposes upon me the burden of mounting a defense and thus constitutes a kind of bureaucratic persecution. I remind you of the statement of Edward R. Murrow when he introduced his now famous T.V. news broadcast repudiating the actions of Senator Joe McCarthy: *"There is a fine line between investigation and persecution."* Like Senator McCarthy's investigators you seem oblivious to the widespread harm you and your committee are causing. I have received many letters from physicians here in Missouri documenting the fact that mine is not an isolated case and supporting my contention that the PRO is incapable of consistently and efficiently discriminating between good and poor medical practice. Think for a minute about the implications of the charges you have made against me: If every nonagenarian suffering from rheumatoid arthritis, bronchiectasis and COPD had to be kept in the hospital until they had normal serum sodium, serum albumin, total protein, consistently sterile urine and consistently normal blood gasses then internists like me will certainly become rich, but the hospitals and our nation will progressively become bankrupt. The federal government through its new DRG system has said it will not pay for such prolonged hospitalizations; now the same government, through you is telling us we must keep people in the hospital longer, order more tests, give more treatments, obtain more consultations and above all document, document, document. It is, thus, no wonder our health care system is so expensive now that watchdogs like you are in charge of holding health costs down. Oh, the glorious irony of it! When I entered practice and docs were controlled by their internal moral compass and by the voluntary ethical restraints imposed by professionalism, health care cost our nation $40 billion. Now, under cost and quality control bureaucrats like you, it costs over $600 billion. When I started medicine no one every heard of wasteful practices like "defensive medicine"; malpractice judgments wee small and rare; the federal fee freeze was unheard of; the DRG system had no yet been invented; no one heard of prospective review, concurrent review or second opinions and doctors were held in high esteem and deserved it. Now a new species of doctor has changed all that. I suppose H. L. Mencken would have called him *boobus medicus*, a sub-species of *boobus bureaucraticus*.

I hope that one day you and your kind will be made answerable for the harm you have caused innocent physicians, patients, the profession and the American health care system. Until that day comes independent practitioners like myself will continue to be harassed, our patients, the profession and the American health care system. until that day comes independent practitioners like myself will continue to be harassed our patients abused, our tax dollars wasted, and the glorious promise of modern health care unrealized.

Physician's Recognition Awards

Seventeen MSMA members qualified for the MSMA-AMA Physician's Recognition Award during December 1991. The award, presented by the Missouri State Medical Association and the American Medical Association, is given to those physicians who voluntarily complete 150 credit hours of continuing medical education within a three-year period. Sixty of the credit hours must be earned in Category 1 programs.

Audrain
Leonard L. Davis, *Hazelwood*

Boone
James A. Shapero, *Columbia*

Cape Girardeau
Carl F. Ritter, *Cape Girardeau*

Greene
D. Davies Foote, *Springfield*
Norman E. Morrow, *Springfield*
David E. Nelson, *Springfield*

Jefferson
S. Sangzin Bai, *Crystal City*

Metro Med
of Greater Kansas City
Nathaniel Winer, *Kansas City*

St. Louis Metro
Dixie Anderson, *St. Louis*
Charles H. Andrus, *St. Louis*
Donald F. Bandle, *St. Louis*
Shael S. Bronson, *St. Louis*
Louis A. Gilula, *St. Louis*
Jonathan A. Gold, *St. Louis*
Virginia M. Herrmann, *St. Louis*
John E. Hironimus, *St. Louis*
Lawrence M. Kotner, *St. Louis*

Thanatology: A New Clinical Specialty to Help Confront Death

Saul Boyarsky, M.D., J.D., Carol S. North, M.D., and Rose E. Boyarsky, Ph.D.
St. Louis, Missouri

When patients are dying, the burden of making health care decisions taxes everyone. This is especially true when patients and doctors must make decisions regarding the timing of the death. The decisions are intensely emotional and far ranging. For the physician, the decisions demand the utmost in clinical skill, knowledge, social acumen and wisdom. The challenge is to turn this inevitable and devastating event into a more positive one.

Within the medical profession, there has been a growing reassessment and debate over the timing of the dying patient's final management. This is exemplified by the Letters to the Editor columns of the *New England Journal of Medicine* dealing with the subject, which are cited at the end of this paper. This debate originally began over the special problems of brain dead patients and "do not resuscitate" orders. It now extends to critical care -- cardiovascular and pulmonary medicine -- where astute physicians are starting to confront death prospectively.

The medical profession has been criticized for being too technologically polarized, failing to show adequate concern for the dying patient. It is true that many physicians avoid issues of death, unable to face them in frank discussions with patients. This avoidance has left dissatisfied patients and families with unfulfilled needs. There is a vacuum of ignorance and fear and an unfair perception that the entire profession has too little empathy and compassion.[1]

The debate over the management of the dying patient has been given impetus by the enactment of the Danforth-Moynihan Patient Self-Determination Act of 1990[2] and the decision by the U.S. Supreme Court in the Cruzan case.[3]

The Supreme Court ruling on the Missouri right-to-die statute in the Cruzan case found a constitutional and common law basis for the right of persons to die in dignity under circumstances of their own choosing, free to refuse artificial sustenance and life support.[3] The decision changed attitudes in this country to favor the use of advance directives (living wills). The full decision has been interpreted to encourage states to pass their own laws governing advance directives and the appointment of proxies and surrogates. This legal clarification has freed doctors to terminate life support in the appropriate case without fear of criminal prosecution. It also encourages patients to designate persons to make medical decisions if they become incompetent, and encourages physicians to honor advance directives.

A New Medical Discipline

When life is ebbing, that transitional stage of existence between life and death -- dying -- is conceptually neither life nor death. There is a need for a set of new principles to provide guidance to better manage the dying process. These principles would formulate the basis for a new discipline, clinical thanatology, a term suggested by Feigenberg and Shneidman in 1979[4]. Clinical thanatology would be defined as the theory and practice of the management of dying.

Clinical thanatology would not be a trivial field. Its complexity can be grasped from an overview of its scientific and theoretical span. Death can occur under some 22 clinical specialties, involving any of eight to 12 organ systems. It may come suddenly or gradually, through a host of different diseases and conditions. Each death has a different clinical scenario. Our diverse technological advances allow an individual to remain alive despite cardiac death, brain death, pulmonary death, or imminent death from burns, cancer or AIDS. It is quite clear why there is a need for a specialty of clini-

> ***Rather than a doctor of death, the medical profession and public need sensitive, learned physicians who can specialize in the problems of dying. This multi-faceted, multi-disciplinary endeavor deserves its own title, clinical thanatology.***

cal thanatology.

Though the counseling functions of the clinical thanatologist would seem to call for a minister, priest or rabbi, possibly a social worker, the selection of a physician for this task would be dictated by the biological and technological complexity of the practice.[7-9] The clinical thanatologist should be a practical, experienced, and broadly educated physician, able to face death reasonably and calmly. This person need not be an expert in all areas but must know where to find experts and what to ask them.

The clinical thanatologist must be a strong leader who is able to forge an effective committee out of an inchoate group of consultants. He must be capable of shepherding a committee of clinicians, family members, ministers, psychologists, lawyers, administrators, and others to consensus. He must conform the decision-making procedure to moral and social guidelines while respecting family values. The clinical thanatologist must act so that the connotations of the word "thanatologist" will contrast sharply with the opprobrium of "Dr. Death."

The Thanatologist's Function

There are a number of ways a clinical thanatologist would function. First, every major medical facility would be advised to have the services of a clinical thanatologist.

Secondly, a clinical thanatologist would anticipate the needs of patients and families, discuss them, educate individuals and help resolve disagreements over the management of the dying patient.

It is clear that the starting point for thanatological decisions are the individual patient's desires, choices and values. The most desirable course, legally, ethically, and medically, is for the individual to state his or her preferences clearly, openly, and for the record in an advance directive. Until now, few people have been this far-sighted and decisive. Oversight, neglect, illness, surprise, and even incompetence interfere with making the decisions required for an advance directive.[5,6] Thus, the third function of the thanatologist would be to help patients write their advance directives.

The option to appoint a proxy from the immediate family with durable power of attorney can serve to clarify the intent of an advance directive and surmount unforeseen problems, such as the development of incompetence. The advance directive would establish the basic principles for an individual to die with dignity. The caretakers could then reject therapeutic measures that are overly heroic. As a fourth function, the thanatologist could guide the proxies and family members should the individual become incompetent.

Prudence has long taught that life and death quandaries are better approached prospectively than retroactively. It is clear that the patient's values and choices for treatment must be stated and recorded in an advance directive. By reconciling the expressed wishes among the patient's several legal documents, the thanatologist could stimulate teamwork between physicians and lawyers as a fifth function.

Clinical thanatology would eventually concern itself with more than the dying patient. Clinical thanatology would enrich and broaden the art and practice of medicine with the principles of ethics, psychiatry, law, medical sociology, and religion to restore a more humanitarian touch to medical practice.

In preference to assisted suicide, we need a sensitive, effective, scholarly clinician, a clinical thanatologist to steer the family and patient through the rocks and shoals of dying. Helping patients decide how to die is not to be confused with deciding who should die, although for some patients, the decisions may overlap.[10]

Summary and Conclusions

The complexities, widespread ramifications and uncertainties surrounding decisions dealing with the process of dying call for a specialty of clinical thanatology. The need for such a specialty grows out of medical technological progress, which has permitted the prolongation of life without parallel guidelines for its termination. The further functions and roles of a thanatologist would evolve with the passage of time. ■

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Invasive Pulmonary Aspergillosis Complicated by Nocardiosis in a Patient with Agnogenic Myeloid Metaplasia

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Among the different *Aspergillus* species, *Aspergillus fumigatus* is most frequently associated with disease. Other species -- including *A. niger*, *A. flavus*, *A. glaucus* and *A. terreus* -- are usually considered contaminants but are also known to cause disease.[1] Identification of a specific *Aspergillus* species is based on the macroscopic appearance of its colony and the microscopic morphologic findings of conidiophores and phialides. Thus, the accuracy of identification depends upon the skill of the laboratory technician. Because of these considerations, some laboratories report suspected isolates belonging to an *Aspergillus* species as being of "fumigatus" or "non-fumigatus" variety.

Though *A. fumigatus* is often associated with disease, as few as 9% of the patients who have this fungus isolated from their sputa have invasive pulmonary aspergillosis (IPA).[2] In patients with leukemia, IPA may be predicted by positive nose cultures for *Aspergillus flavus*.[3] Clinical correlation is extremely important in assessing the pathogenicity of an *Aspergillus* isolate in an individual patient. In this case report, IPA evidenced by a rapidly growing solitary lung mass, was undiagnosed and untreated because the *Aspergillus* species was classified as a "non-fumigatus" variety. The invasive disease was later complicated by pulmonary and pleural nocardial infection.

Case Report

A 73-year-old white man was hospitalized in June 1989 with a two-month history of progressive shortness of breath, easy bruisability and weight loss. He had significant anemia (Hb=6.8 g/dL), leukocytosis (16,600/mm^3) and thrombocytopenia (platelets=86,000/mm^3). Symptoms resolved with transfusion of six units of packed red blood cells. Bone marrow biopsy established a diagnosis of agnogenic myeloid metaplasia. Splenomegaly was present. He was treated with prednisone and ethylestrenol. Two months later, he was hospitalized for a blood transfusion. Admission chest roentgenogram revealed a mass in the right upper lobe (Fig 1A). The chest roentgenogram obtained at the other hospital two months earlier was not available for review. A computed tomographic scan of the chest confirmed the mass and also revealed bullous lung disease (Fig 1B). Spirometry revealed reversible airflow obstruction, and bronchodilator therapy was initiated. The patient had no respiratory complaints and remained afebrile. A fiber optic bronchoscopy was performed, revealing normal endobronchial anatomy. Brushings and washings were obtained from the right upper pulmonary lobe. Acid fast bacilli (Zeihl-Neilsen stain) and fungi (Giemsa stain) were not detected on the smears of the bronchoscopic specimen, and bacterial culture of the bronchial washings revealed normal oral flora. Four days later, *Aspergillus* of "non-fumigatus" variety was isolated from the bronchial washings. *Aspergillus* "non-fumigatus" was also isolated from two sputum specimens obtained prior to the bronchoscopy. Eight days later, a chest roentgenogram showed the right upper lobe mass had enlarged (Fig 2A), but unfortunately, by then the patient had been discharged.

Fifteen days after the initial *Aspergillus* identification, the patient was admitted to another local hospital with complaints of increasing shortness of breath and cough with hemoptysis. He was treated with intravenous gentamicin and piperacillin for two weeks without improvement. Following discharge, he was readmitted a week later to the same hospital with worsening of previous symptoms. Chest roentgenogram revealed alveolar infiltrates in the right lung and a new right pleural effusion. Two days later (45 days since initial isolation of *Aspergillus*), he underwent intubation and mechanical ventilation for respiratory failure and was transferred to the University Hospital. Chest roentgenogram showed consolidation of the right lung (Fig 2B). Bronchoscopy revealed bleeding and stringy material coming out of the right lower lobe bronchus. Bronchoalveolar lavage was not diagnostic after Giemsa, Zeihl Nielsen stains, and cytologic examination of the sediment. Thoracentesis revealed the pleural effusion to be a transudate without organisms.

Open lung biopsy (right upper and middle lobes) was performed the next day. The specimen showed minimal interstitial mononuclear inflammation and a variable degree of interstitial fibrosis. Gomori-methenamine silver and Zeihl-Nielsen stains did not reveal any pathogen. Amphotericin B therapy was started on the day of transfer. The patient received 70 mg of amphotericin B over the next four days. Administration of the drug was then stopped and trimethoprim-sulfamethoxazole (5 mg/kg every six hours) administered because organisms compatible with *Nocardia* were noted in the Gram stain of the sputum. Three days later amphotericin B therapy was restarted. The patient's pleural effusion was tapped again on the 12th day of this hospitalization. On this occasion, the fluid was exudative, with few leukocytes (400/mm^3), predominantly neutrophils (94%).

Administration of trimethoprim-sulfamethoxazole (for a total of 11 days) and amphotericin B (cumulative dose 580 mg) were continued until the patient's death on day 16. After his death, fungal cultures of both bronchoalveolar lavage and open lung biopsy specimens revealed growths of *Aspergillus fumigatus*. *Nocardia asteroides* was isolated from the second sample of the pleural fluid and from two sputum samples obtained on the fifth and 15th day of the second hospitalization. Autopsy revealed disseminated invasive aspergillosis involving the heart, thyroid, liver, pancreas, peri-adrenal fat, kidneys, and right lung. *Nocardia* organisms were neither identified nor cultured from the necropsy specimen.

Discussion

Aspergillosis is frequently suspected in a febrile neutropenic patient with a hematologic/lymphoreticular malignancy, especially when administration of broad spectrum antibiotics has been ineffective. Amphotericin B therapy is recommended in these cases while attempts are made to culture the fungus from respiratory secretions or to find the characteristic fungal hyphal invasion in the lung tissue. It is of interest that two large studies found that 20% of patients with IPA had normal neutrophil counts.[2,4]

Although agnogenic myeloid metaplasia is a myeloproliferative disorder, it has not been associated with IPA in the absence of leukopenia. Only one of 98 patients in Young's series had agnogenic myeloid metaplasia,[4] but this patient was also neutropenic.

Chest roentgenograms may be normal in up to a third of patients with IPA. Otherwise, the films reveal bilateral nodular densities suggestive of septic pulmonary emboli or patchy pneumonitis, which is slowly progressive.[4] It is not unusual for a single nodule to be the presenting radiographic abnormality in invasive pulmonary aspergillosis.[5,6] Albeda et al noted that 23 of 26 patients with IPA complicating leukemia and neutropenia had either single or multiple pulmonary nodules.[5] Nodular infiltrates usually appeared 17 days after the onset of chemotherapy-induced granulocytopenia. These nodules enlarged slowly as other nodules appeared in other parts of the lung.

Bone marrow recovery markedly affects the clinical and radiographic course of IPA. Among the 15 patients whose bone marrow recovered, none developed diffuse lung disease, but cavitation of the pulmonary nodules occurred in 73% (usually two days after the granulocyte count was greater than 500/mm^3). Of the 11 patients with persistent leukopenia, diffuse lung disease developed in

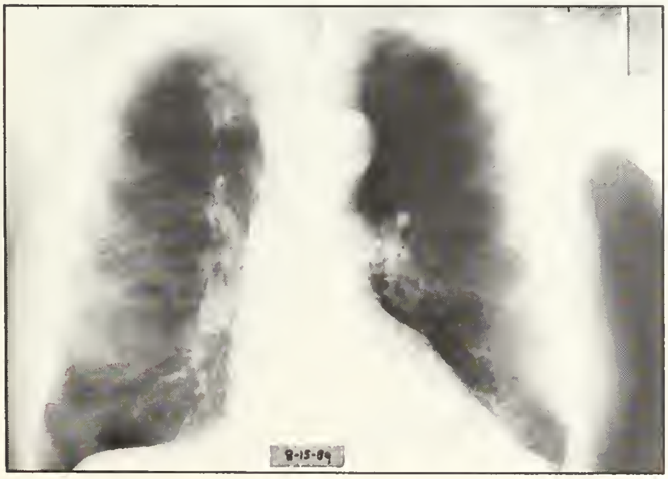

Figure 1-A
Chest roentgenogram

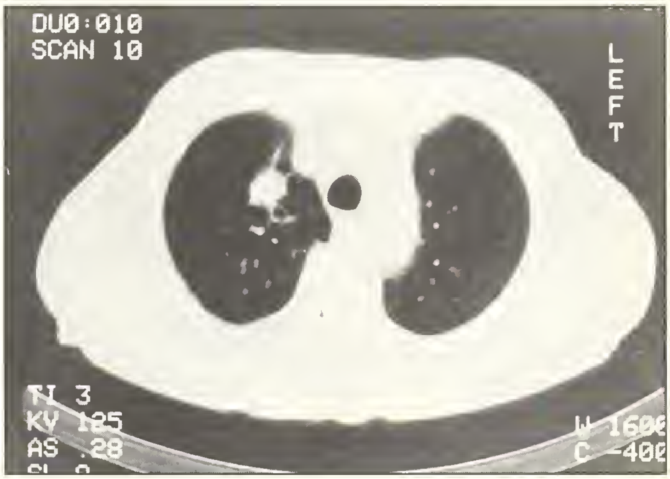

Figure 1-B
Computed tomography of chest showing a mass in the right upper lobe.

73% and cavitation occurred in none.

The pattern of progression of radiographic abnormalities of IPA in a host with normal leukocyte counts is not well defined. In our patient, the left lung was found to be uninvolved at autopsy, despite dissemination of disease to other organs. A typical sign of *Aspergillus* growth (aspergilloma) in the lung cavity is thickening of the overlying pleura.[7] It is not known whether this is caused by actual fungal invasion of the pleura or by an immunologic reaction. Although rare, *Aspergillus* infection of the pleural space can occur as a complication of pulmonary tuberculosis, preexisting chronic empyema, bronchopleural fistula, contamination of the pleural space during lung resection, thoracentesis, or therapeutic pneumothorax.[8] Pleural invasion in IPA is extremely unusual.[4]

In our patient, pleural effusion was not due to *Aspergillus* invasion. Pleural effusion was noted on the chest radiograph during the second hospitalization (Fig 2B). Thoracentesis revealed this effusion to be a transudate. Infectious causes were excluded by appropriate cultures. Twelve days later, however, (after the patient had received 230 mg of amphotericin B and six days of intravenous trimethoprim-sulfamethoxazole therapy) thoracentesis revealed a change in the character of the fluid and cultures confirmed that the effusion was due to *Nocardia asteroides* infection. At necropsy, *Nocardia* was not noted in the pleura of either lung, indicating successful eradication of the organism prior to the patient's death.

In immunosuppressed patients, isolation of *Nocardia asteroides* from the sputum may indicate infection or colonization.[9,10] *Nocardia asteroides* was isolated from both sputum and pleural fluid in our patient, ruling out colonization. We are not aware of any previous report of nocardiosis complicating IPA. This might be suspected in a patient with IPA who develops a pleural effusion.

Invasive pulmonary aspergillosis should be suspected when any *Aspergillus* species is isolated from a respiratory specimen of a patient with an abnormal chest roentgenogram, particularly in an immunosuppressed patient. Therapy should be initiated promptly. In our patient, it was unusual that even an open lung biopsy did not document IPA histologically. This again underscores the importance of tissue culture when aspergillosis is suspected. Inability to find fungal tissue invasion in an open lung biopsy specimen does not exclude IPA, and this finding alone should not determine the course of therapy.

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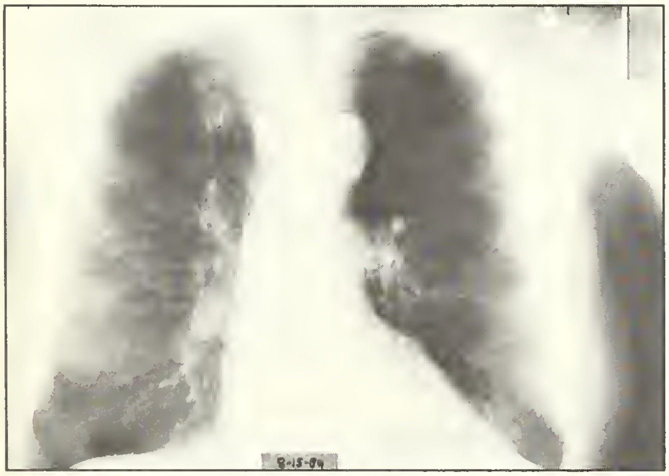

Figure 2-A
Chest roentgenogram done 8 days later showing a rapid growth of the mass lesion in the right upper lobe.

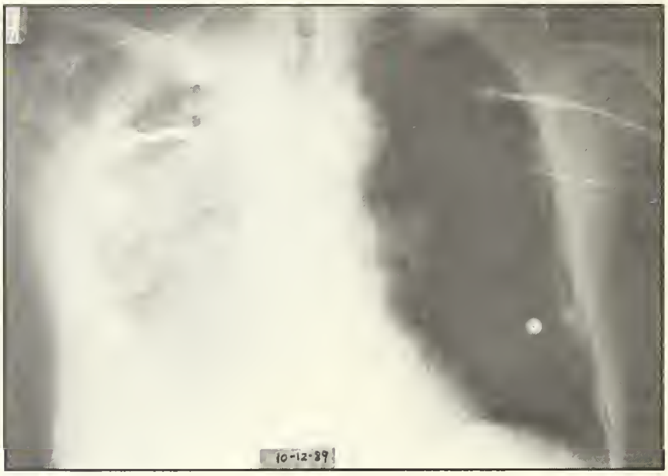

Figure 2-B
Chest roentgenogram done 58 days later showing diffuse consolidation of the right lung and right pleural effusion.

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Seat Belt Usage by Trauma Center Personnel

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Properly worn automobile safety belts may prevent over 40% of front seat auto occupant fatalities, yet only 19-23% of Americans wear them voluntarily.[1-4] In July 1984, New York state set a precedent by becoming the first state to adopt legislation requiring motorists driving in or through New York to wear safety belts. The law requires motorists and all front seat passengers over 4 years of age to wear auto restraints. Children under 10 years of age, regardless of their location in the vehicle, are required to be restrained, and those under 4 years must be in an approved child restraint device. Violators are subject to civil fines of up to $50 with drivers being financially responsible for their own fines and the fines for occupants under 16 years of age; passengers older than 16 are responsible for their own fines. In its first six months in effect, this law is estimated to have averted 220 fatal, 1,500 severe, 4,600 moderate and 2,600 light injuries across the state.[1,2]

In 1984, the U.S. Department of Transportation issued Federal Motor Vehicle Safety Standard 208, which phased in the required installation of passive restraints (automatic safety belts or air bags) in all new cars by the 1990 model year.[4] In 1985, Missouri passed Senate Bill 43, which stated that by 1987, all front seat occupants were to be secured by a seat belt. In an effort to evaluate the compliance of this bill at a Missouri Level I Trauma Center, a prospective study was carried out.

Automobile seat belt usage decreases morbidity and mortality. However, despite a Missouri state law mandating seat belt usage, only 54% of front seat occupants in automobiles are secured by a seat belt. A study of health care personnel at a St. Louis Level I Trauma Center found a compliance rate for front seat occupants of 73%. Although this is not total compliance, health care workers appear to recognize not only the law but the safety of seat belt restraints.

Material and Methods

To evaluate seat belt usage by health care personnel at a Level I Trauma Center for the states of Missouri and Illinois, the Division of Trauma Services at St. Louis University Medical Center monitored seven parking lots and structures in which University Hospital health care workers parked. Surveillance was done from 6:00 a.m. through 8:30 a.m. on a clear Tuesday, May 21, 1991. There was no warning about this audit, nor were there any seat belt safety sessions held, other than at an annual health fair held eight months earlier. All vehicles entering the lots were stopped, and drivers and passengers were observed to see whether they were wearing their seat and shoulder belt harnesses.

In addition, as part of an educational effort, the health care workers were given information sheets on the Missouri seat belt law, and had "Buckle Up" stickers placed on their lapels. If the car's occupants were in compliance, a blue ribbon was tied on the car door handle or radio antenna. Those in compliance also received donated incentive gifts. Those not in compliance were informed about the incentives they missed and educated on automobile restraint usage.

Results

Six hundred and ninety-six automobiles were inspected. Five hundred and nineteen (75%) drivers were wearing their seat and shoulder belt. Forty-four automobiles had front seat passengers, of which 23, or 52%, were wearing seat restraints. Thus, the total occupant compliance rate was 542 of 740 (73%). The highest compliance rate was in the parking lot occupied by physicians -- with 82 of 102 (80%) wearing a seat belt, followed by the parking lot occupied by non-nurses and non-physicians -- with 199 of 260 (75%) compliant. Finally was a parking area predominantly used by nurses, with 238 of 328 (73%) compliant.

Discussion

In the past 100 years, approximately 2 million persons have died and nearly 100 million have been injured from vehicular trauma. Between 1984 and the year 2000, it is expected that at least 6 million more will die and 350 million will be injured worldwide.[3] It is estimated that in the United States every 10% increase in safety belt use saves about 1,500 lives, more than 30,000 moderate to serious injuries, and about $800 million in direct costs.[4] After implementation of its mandatory seat belt law, North Carolina has seen a significant reduction in severe and fatal injuries among front seat car occupants who are involved in crashes.[6] In metropolitan Chicago, it has been shown that seat belt wearers had a 60% reduction in severity of injury, a 65% decrease in hospital admissions and 66% decline in hospital charge when compared with non-users of safety belts.[7]

Presently in the United States, 37 states and the District of Columbia have some form of seat belt legislation.[5] Overall, seat belt usage is estimated to be at approximately 46%.[4] When New York began enforcing its law on January 1, 1985, seat belt usage in New York increased from 21% to 63%. This is similar to trends observed in other countries, including Canada and (West) Germany. However, a follow-up study in New York showed a drop to 37% compliance in 1986, a statistically significant decrease.[1,8]

As mentioned, Missouri's seat belt law went into effect in 1987 for all front seat occupants. However, the law dictates that the unrestrained occupant be cited only after he or she has been stopped for another offense. The fine is $10.[5] The state of Missouri has a 54% seat belt usage rate, with the highest usage rates approximately 60% in the metropolitan areas of Kansas City and St. Louis.[5] The 73% compliance rate we found among health care workers at a major St. Louis trauma center perhaps reflects a group more educated with regard to health safety. The rate may also reveal the facility's metropolitan location. The fact that the non-nurse/non-physician personnel had a high compliance rate is interesting and may reflect a more motivated and interested group of individuals in preventive health care measures.

There is little debate that the use of seat belts decreases morbidity and mortality in car crashes. However, health care providers, in addition to using seat belts themselves, should continue to educate not only their families but also their friends on the importance of seat belt usage. Auto safety eduction begins with prevention. Health care workers at a metropolitan trauma center demonstrated their use of seat belts, although one-fourth of the drivers were not restrained. All health care providers, whether working in a trauma center or not, should serve as models, following the law and helping to educate the public. Seat belts do save lives. The best educators are the survivors of major crashes, those who wear safety belts, and those who care for victims of crashes.

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The authors also wish to acknowledge the participation and support of the following members of the trauma team: Mary Filer, LPN; Lee Foster, MSW; Sally Frese, R.N.; Shari Helbig, R.N.; Karen Mikhail, R.N.; Connie Pilat, R.N.; Mary Beth St. John, R.N.; Nancy Toy, R.N.

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Recent Trends in the Selection and Fitting of Hearing Aids

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A large segment of the hearing impaired population does not utilize amplification. this paper reviews several factors which may account for the under-utilization of hearing aids by the hearing impaired population. In addition, this paper reviews several recent developments in procedures used to select and fit hearing aids.

In a previous paper,[1] it was suggested that 18 to 22 million people in the United States have hearing loss that could be helped by hearing aids.[2] It has also been reported that only 4 million of these people are currently wearing hearing aids.[2] There are many explanations as to why 14 to 18 million people who could benefit from hearing aids are not currently using amplification.

For instance, primary care physicians may believe that the reported hearing loss is a "minor problem" and does not warrant further investigation,[3] and thus fail to make the referral. In addition, there are primary care physicians and specialists who believe hearing aids will not help patients with sensorineural hearing loss (e.g., "nerve deafness"). On the other hand, potential hearing aid users believe that hearing aids "amplify everything" and provide little benefit when listening in a noisy environment. They may also believe their communication difficulties are "not that bad" and they "hear OK," and that their communication problems could be resolved without a hearing aid if only "people would talk louder and not mumble."

One key to improving poor hearing is an appropriate referral for amplification from the patient's physician. Unfortunately, some physicians may be misinformed about the benefits of hearing aids provide to individuals with sensorineural hearing loss (nerve deafness) and may be unaware of significant improvements in the way hearing aids are fitted and selected.

We review recent developments in the selection and fitting of hearing aids. This information should help physicians understand the benefits of newer hearing aids, enabling them to counsel patients appropriately.

Advances in the Selection and Fitting of Hearing Aids

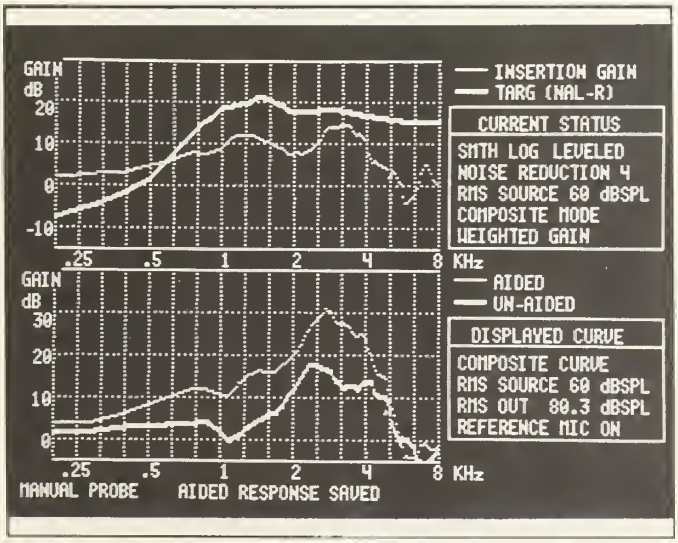
Two things are needed for a successful hearing aid fit. The first is calculation of the gain required to amplify conversational speech in specific frequency regions to a loudness level judged by the patient to be comfortably loud. Second is assurance that the output of the hearing aid (e.g., intensity level of the signal at the input to the microphone, plus the gain) does not exceed a loudness level judged by the patient to be uncomfortably loud or irritating.

Until recently, hearing aid dispensers had no clinically useful tools to determine appropriate gain and output for their clients. Few knew the appropriate amount of gain and output required at each frequency to compensate for the hearing loss and changes in growth of loudness that usually accompany sensorineural hearing loss. Hearing aids were commonly dispensed with the simple goal of demonstrating that aided performance was better than unaided performance. It was not general practice to adjust the hearing aid settings so that the measured aided performance matched the prescribed aided performance. Furthermore, there was little emphasis on adjusting the aid for the best everyday hearing. With this in mind, it is not surprising that patients did not benefit from amplification and told friends and relatives of their dissatisfaction with hearing aids.

Prescriptive Fitting

Until recently, it was not uncommon for hearing aid dispensers to verify a fitting by simply placing the hearing aid in the ear canal and asking the patient if it "sounded OK." Unfortunately, this type of hearing aid fitting is still common today. With this method, no clear cut decisions are made regarding the appropriate amount of gain or output for the patient. Clearly, this is a rather "unscientific" manner in which to select and fit hearing aids, prone to provide an inadequate fit. It is little wonder these hearing aids end up in a drawer and patients complain of little benefit.

In the past decade several prescriptive procedures, based on the evaluation of large groups of subjects, have been developed to guide the dispenser in prescribing the gain required to amplify average conversational speech to the patient's most comfortable loudness level. For example, one prescriptive rule suggests that the prescribed gain in specific frequency regions should approximate half the hearing loss (e.g., the POGO prescriptive rule of McCandless and Lyregaard[4]), while another rule suggests gain should be one-third or two-thirds depending on the degree of the hearing loss.[5] Still another prescriptive rule suggests gain should be between a third to a half of the hearing loss (e.g., the NAL-R rule of Byrne and Dillon[6]). Although these prescriptive procedures vary significantly in prescribed gain, they provide an initial fitting target that may be achieved and then modified to meet the individual's amplification needs. The process results in more satisfied hearing aid users. Simply put, the availability of these prescriptive procedures provides for a more "scientific" approach to the selection and fitting of hearing aids.



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CURRENT STATUS
SMTH LOG LEVELED
NOISE REDUCTION 4
RMS SOURCE 60 dBSPL
COMPOSITE MODE
WEIGHTED GAIN

DISPLAYED CURVE
COMPOSITE CURVE
RMS SOURCE 60 dBSPL
RMS OUT  80.3 dBSPL
REFERENCE MIC ON

MANUAL PROBE    AIDED RESPONSE SAVED
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Figure 1: Prescribed NAL-R real ear insertion gain as it appears on the monitor of a real ear analyzer.

Real-Ear, Probe-Tube Microphone Measures

Concurrent with the development of prescriptive procedures was the introduction of real-ear, probe-tube microphone measures to quickly and reliably verify whether the prescribed gain is received by the listener. In the past, the usual method to verify the hearing aid fit was functional gain measures. The dispenser measured the difference between the unaided and aided sound-field thresholds for discrete frequencies between 250 to 4000 Hz in octave and mid-octave intervals. The procedure has several limitations.[7] For example, functional gain is measured only at discrete octave and mid-octave frequencies, but the real-ear gain of a hearing aid may have numerous resonances and antiresonances between these frequencies. Therefore, real-ear performance of the hearing aid at non-test frequencies is not measured. Second, there is more test-retest variability of functional gain measures than with probe-tube microphone measures. The differences between repeated functional gain measures must be considerable in order for the dispenser to observe them. Third, the patient must make overt responses to establish

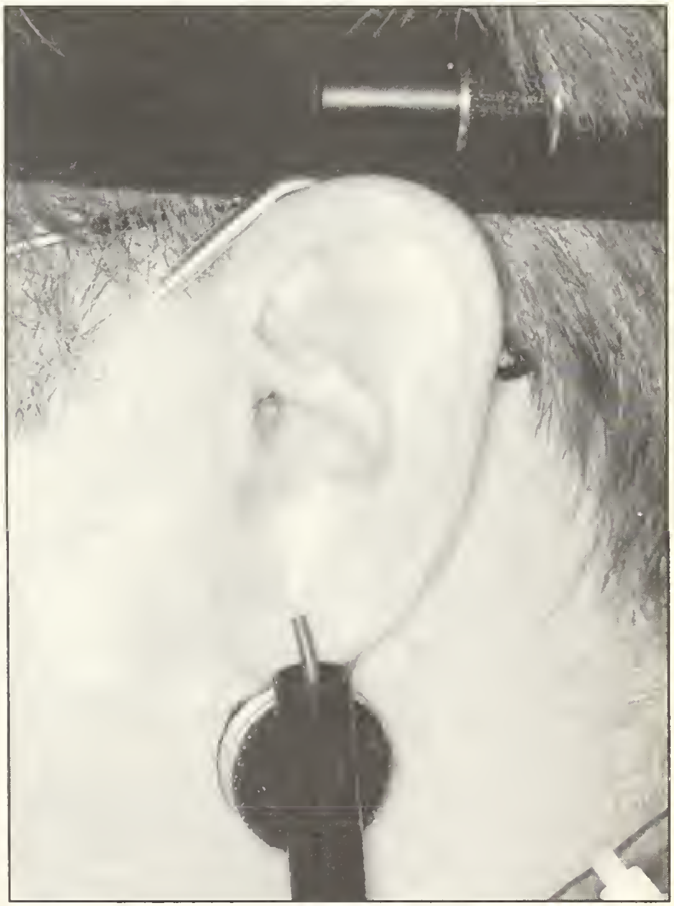
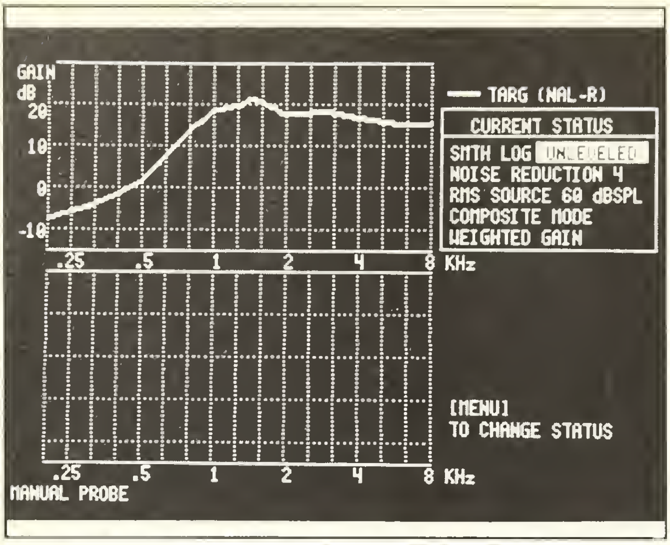


Figure 2: Placement of the reference (upper) and probe microphones (lower) for real ear measures. Notice the mark on the silicone tube for placing the probe tube at the tragal notch.



CURRENT STATUS
SMTH LOG UNLEVELED
NOISE REDUCTION 4
RMS SOURCE 60 dBSPL
COMPOSITE MODE
WEIGHTED GAIN

[MENU]
TO CHANGE STATUS

Figure 3: Measured real ear responses appearing on the monitor of a real-ear analyzer. *Lower graph*: solid line represents the REUR; thin line represents the REAR. *Upper graph:* solid line represents the prescribed NAL-R REIR; thin line represents the measured REIR.

the unaided and aided sound-field thresholds. Therefore, functional gain measures has limited use in evaluating children or other patients who may not be able to respond overtly. Finally, the gain of the hearing aid may be underestimated in the lower frequencies due to the presence of ambient noise which, when amplified, may mask the low frequency signal.

An increasingly popular method of verifying real ear benefit from amplification is the recent introduction of real-ear probe-tube microphone analyzers. This method has proven to be efficient and reliable.[1,8] In addition, probe tube measures are objective and responses are not required from the patient. Real-ear gain can be measured at all test frequencies between 100 Hz to 10,000 Hz.

Briefly, with this procedure, audiometric thresholds are entered in the real-ear analyzer, which then generates a prescribed real-ear insertion gain "target" (e.g., POGO, NAL-R, or 1/3). The "target" typically appears as a solid line on the analyzer's monitor (Fig 1). In this example, the prescribed NAL-R real-ear insertion gain is approximately 18 dB at 1000 Hz, falling to approximately 15 dB at 8000 Hz.

Two microphones are usually placed on the patient (Fig 2). The reference microphone (upper microphone) is typically placed over the pinna or at the cheek. This microphone monitors the intensity of the signal (pure-tones, narrow band noise or speech weighted noise) to assure that the signal arrives at a constant intensity at all frequencies (plus or minus 2 dB) at the microphone of a behind-the-ear hearing aid. A soft silicon tube from the probe microphone (lower microphone) is placed in the entrance of the ear canal until the tip is approximately 4-6 mm from the eardrum. This placement assures accurate and reliable real ear measures through 3000 Hz.

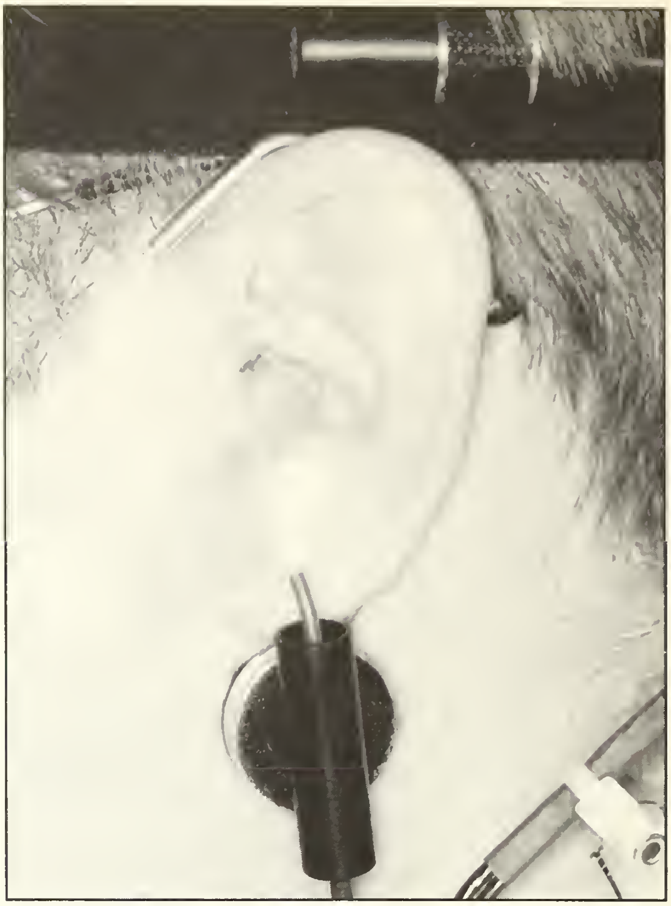


Figure 4: Placement of a ITE hearing aid in the ear canal to measure the REAR and REIR response.

At this point, a signal (usually 60-70 dB SPL of speech weighted composite noise) is emitted from a loudspeaker placed approximately 18 inches from the listener. The real-ear analyzer then measures the intensity of the signal arriving at the eardrum without the hearing aid in place (real ear unaided response - REUR). Figure 3 shows this measurement, the solid curve in the bottom graph.

With the hearing aid placed in the outer ear, the real ear aided response (REAR) is measured with the probe tube microphone (Fig 4). The response is represented by the upper thin curve in the bottom graph of Figure 3.

The analyzer program calculates the measured real ear insertion response (REIR) -- the actual gain provided by the hearing aid -- by subtracting the real ear unaided response (REUR) from the real ear aided response (REAR). The measured REIR is represented by the lower thin curve in the top graph of Figure 3.

The hearing aid dispenser determines whether or not the measured and prescribed REIR agree. If the two responses are in close agreement, then the hearing aid is providing the prescribed amount of gain. If there are significant differences between the responses, then the dispenser can alter the frequency/gain response to more closely match the prescribed target. In the example shown, the measured REIR is considerably below the prescribed REIR at most frequencies. Most dispensers would consider this an unacceptable fit and find ways to achieve greater overall gain. This may include changing the earmold or tubing design, reducing the maximum output, or introducing compression if this were a behind-the-ear (BTE) fitting. If this were an in-the-ear (ITE) or in-the-canal (ITC) fitting, the dispenser could also request a different circuit design from the manufacturer.

Conclusion

Until recently, hearing impaired persons have been hesitant to seek the benefits of amplification. This hesitancy may be due, in part, to inappropriate hearing aid fittings of the past, when there were no valid and reliable tools to accurately "prescribe," verify, and modify fittings as necessary. In addition, the hearing aid technology of the past provided amplified signals that were loud, but not as clear as those available today.

The availability of established prescriptive fitting formulas, the ability to adjust the hearing aid output using real ear probe-tube measures, and the further adjustment of the hearing aid to meet individual needs have had a significant impact on the successful fitting of hearing aids.

Today, greater accuracy is possible in prescribing and verifying hearing aid fittings. In addition, many current hearing aids provide a clearer amplified signal for more effective listening in both quiet and noisy environments.

At one time, the criterion for determining who was a candidate for amplification centered on the magnitude and configuration of the hearing loss. With current technology, any patient reporting difficulties in understanding speech is a candidate for amplification. This includes patients with hearing loss limited to frequencies at 3000 Hz and above.

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In Memoriam

■ **Donald E. Eggleston, M.D.**, Macon, Missouri, died September 27, 1991 at the age of 89. Born in Kingman, Kansas, Dr. Eggleston attended Kansas University and received his medical degree from Washington University School of Medicine-St. Louis in 1930. Dr. Eggleston was an eye, ear, nose and throat specialist. He was a member of the American Medical Association and the Missouri State Medical Association.

■ **William E. Taylor, M.D.**, Springfield, Missouri, died December 10, 1991 at age 83. Born in Springfield, Dr. Taylor attended Teachers College there, and went on to receive his medical degree from Rush Medical College in Chicago in 1935. Specializing in internal medicine, he served as the Health Director of Teachers College, and on the staff at Burge Hospital.

■ **James W. Owen, Jr., M.D.**, Bartlesville, Oklahoma, died December 16, 1991 at age 67. He earned his bachelor's and medical degrees from Washington University, and served in the Army Medical Corps in World War II. During his 40 year career, Dr. Owen worked in private practice before serving as a faculty member at Washington University School of Medicine. He later returned to private practice in Bartlesville before returning to St. Louis to serve as a medical director for the Jewish Center for the Aged until he retired in 1986. A past secretary of the St. Louis Metropolitan Medical Society, Dr. Own was also a member of the St. Louis Internist Club, the Missouri and Oklahoma State medical societies, the American Society of internal Medicine and the Alpha Omega Alpha Honorary Medical Society.

■ **George A. Cowan, M.D.**, Kansas City, died December 3, 1991 at age 72. Dr. Cowan was born in Scotland and received his medical degree from the Glasgow University Medical School in 1944, before joining the British Royal Army Medical Corps. He then served in the Malayan Medical Service and studied at the Institute for Medical Research, Kuala Lumpur, Malaysia. He later instructed at the University of Saskatchewan in Saskatoon before becoming a professor of therapeutic radiology at the University of Florida, Gainesville in 1963. Dr. Cowan was professor of therapeutic radiology at the University of Kansas Medical Center from 1965 to 1970, and then continued in private practice in Kansas City until his retirement in 1984. He was a member of the Metropolitan Medical Society of Greater Kansas City.

■ **Lyle B. Knight, M.D.**, Lee's Summit, died December 7, 1991 at the age of 86. Dr. Knight attended the University of South Dakota in Vermillion and received his medical degree from the University of Illinois in Chicago in 1931. After practicing in Maysville, Missouri, Dr. Knight opened private practice in Lee's Summit in 1936, and co-founded the Lee's Summit Clinic in 1959. He was a staff physician at St. Luke's Hospital for 30 years and was an honorary staff member for 25 years after his retirement in 1966. Dr. Knight was a charter member of the American Academy of Family Physicians and received its Family Physician of the Year award in 1966. He was a member of the American, Missouri, and the former Jackson County medical societies.

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[1] Data on file, Lilly Research Laboratories.

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nizatidine capsules

Brief Summary. Consult the package insert for complete prescribing information.

Indications and Usage: 1. *Active duodenal ulcer*–for up to 8 weeks of treatment at a dosage of 300 mg h.s. or 150 mg b.i.d. Most patients heal within 4 weeks.

2. *Maintenance therapy*–for healed duodenal ulcer patients at a dosage of 150 mg h.s. at bedtime. The consequences of therapy with Axid for longer than 1 year are not known.

3. *Gastroesophageal reflux disease (GERD)*–for up to 12 weeks of treatment of endoscopically diagnosed esophagitis, including erosive and ulcerative esophagitis, and associated heartburn at a dosage of 150 mg b.i.d.

Contraindication: Known hypersensitivity to the drug. Because cross sensitivity in this class of compounds has been observed, H_2-receptor antagonists, including Axid, should not be administered to patients with a history of hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*–1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests–False-positive tests for urobilinogen with Multistix® may occur during therapy

Drug Interactions–No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility–A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups.The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy–Teratogenic Effects–Pregnancy Category C–Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers–Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use–Safety and effectiveness in children have not been established.

Use in Elderly Patients–Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic–Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular–In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS–Rare cases of reversible mental confusion have been reported.

Endocrine–Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely

Hematologic–Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental–Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity–As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other–Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

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Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

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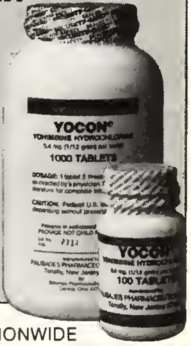

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Student Loans and Moonlighting

by C. Christopher Pittman, M.D.
Chairman, MSMA-RPS Governing Council

A 1983 AMA national survey showed that half of third-year residents moonlight. A 1987 *Journal of Family Practice* study found more than two-thirds of second- and third-year family practice residents moonlight. Moonlighting by resident physicians is not new, however. A recent survey of doctors trained from the 1930s to the 1950s revealed almost one-third moonlighted.

Today, for many residents, moonlighting has become a necessity. The Federal government has curtailed deferment of student loans for the duration of training to a maximum of two years after leaving medical school. Student loan interest is no longer tax deductible. To my knowledge, there are no post-graduate programs which require less than three years of training. The Association of American Medical Colleges reports three-fourths of all medical graduates owe money for their education, with an average debt of over $45,000. Many, including several of my closest friends, owe far more upwards of $75,000 to $100,000.

Loss of loan deferment and forbearance will undoubtedly affect new physicians' choice of specialty, practice location and private versus academic practice setting. New residents with high debt burdens will less likely enter primary care specialties or practice in rural or under-served areas. We are losing talented potential academicians to more lucrative private practice opportunities, and many residents are foregoing fellowships in order to realize needed income.

Residency and fellowship programs are lengthening every few years. Most gastroenterology and cardiology fellowships, after a minimum three-year internal medicine residency, are three years or even four years if research is required or if one has a desire to learn special interventions. A typical neuro-radiologist will have spent seven years in postgraduate training: one year internship, four years residency and two years fellowship. Many residents and fellows also must support families during this long and arduous training process. The stress of debt obligations has contributed to the deterioration of the family life of residents and resulted in some divorces.

These are a few of the reasons why the AMA Resident Physician Section has declared student loan deferment its top priority for 1992. Congress is considering proposals that would kill deferment and eliminate forbearance as well. The AMA has responded by agreeing to mobilize a nationwide lobbying coalition.

I do not want to imply that moonlighting is itself wrong ... just that many residents have no choice. The inherent rewards of moonlighting include money, of course, for student loans, family needs, and expensive medical texts. The additional income may also springboard one to learn more about personal finance and money management. Moonlighting is an opportunity to mature as a physician and may well expose certain skill and knowledge deficits which will hopefully provide impetus to study and improve. Moonlighting often provides exposure to a broader patient population, helps you master additional skills, and develops self-confidence.

SOME ADVICE:

- Never allow compromise of patient care at your training program or your moonlighting job due to fatigue or overwork. Know your limitations.
- Become familiar with the types, various coverages, and limits of malpractice insurance and never practice without it!
- Never take on responsibilities your training has not prepared you for, or at least have access to consultants.
- Most important, moonlighting IS practicing medicine, and that, in itself, certainly has merit.

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New Members

Susy Alias, M.D., 15 Maryhill, St. Louis, MO 63124, has become a member of the Missouri State Medical Association. Dr. Alias received her baccalaureate degree from Little Flower College, Guruvayur, S. India and obtained her medical degree from the University of Kerala, S. India in 1969. She completed her residency at the University of Missouri-Columbia Hospitals & Clinics. Dr. Alias specializes in physical medicine and rehabilitation.

James S. Appelbaum, M.D., 4320 Wornall, Suite 332, Kansas City, MO 64111, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Appelbaum received his baccalaureate degree from the University of Kansas-Lawrence and obtained his medical degree from the University of Kansas School of Medicine, Kansas City in 1986. He completed his residency at the University of Chicago Hospitals, Chicago, Illinois. Dr. Appelbaum specializes in neurology.

David L. Black, M.D., 2817 McClelland Blvd., Suite 125, Joplin, MO 64804, has become a member of the Jasper County Medical Society. Dr. Black received his baccalaureate degree from Pittsburg State University, Pittsburg, KS and obtained his medical degree from the University of Kansas School of Medicine, Lawrence-Kansas City in 1984. He completed his residency at Kansas University Medical Center. Dr. Black specializes in orthopaedic surgery.

Kevin J. Blinder, M.D., One Hospital Drive, Columbia, MO 65212, has become a member of the Boone County Medical Society. Dr. Blinder received his baccalaureate and medical degree from the University of Missouri-Kansas City in 1985. He completed his residency at the University of Missouri-Columbia Hospitals & Clinics. Dr. Blinder specializes in ophthalmology.

Paul E. Callicoat, Jr., M.D., 2817 McClelland, Suite 128, Joplin, MO 64804, has become a member of the Jasper County Medical Society. Dr. Callicoat received his baccalaureate degree from the University of North Carolina at Wilmington and obtained his medical degree from East Carolina University School of Medicine, Greenville in 1985. He completed his residency at St. Lukes Hospital, Kansas City, Missouri. Dr. Callicoat specializes in cardiology.

Susanne M. Chow, M.D., 8900 Indian Creek Pkwy., #450, Overland Park, KS 66210, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Chow received her baccalaureate degree from Pittsburg State University, Pittsburg, KS and obtained her medical degree from the University of Kansas Medical School, Kansas City in 1985. She completed her residency at the University of California-Los Angeles. Dr. Chow specializes in diagnostic radiology.

Terrence W. Coleman, M.D., 1515 W. Truman Road, Suite 403, Independence, MO 64050, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Coleman received his baccalaureate degree from Harvard University, Cambridge, Massachusetts and obtained his medical degree from Albany Medical College, Albany, NY in 1979. He completed his residency at Wilford Hall USAF Medical Center. Dr. Coleman specializes in gastroenterology.

Joseph E. Conrace, D.O., 2817 McClelland, Suite 212, Joplin, MO 64804, has become a member of the Jasper County Medical Society. Dr. Conrace received his baccalaureate degree from the University of Virginia, Charlottesville, VA and obtained his medical degree from the University of Health Sciences College of Osteopathic Medicine, Kansas City in 1968. He completed his residency at Western Missouri Mental Health Center, Kansas City, MO. Dr. Conrace specializes in adult and child psychiatry.

Kent A. Crighton, M.D., 90 Gravel St., Cassville, MO 65625, has become a member of the Ozarks Medical Society. Dr. Crighton received his baccalaureate degree from Drury College, Springfield, MO and obtained his medical degree from Ross University, Portsmouth, Dominica in 1987. He completed his residency at Deaconess Hospital, St. Louis, MO. Dr. Crighton specializes in internal medicine.

Donald J. Curran, D.O., 1000 W. 10th St., Rolla, MO 65401, has become a member of the Mid-Missouri Medical Society. Dr. Curran received his baccalaureate degree from Texas A & M University, College Station and obtained his medical degree from the University of Health Sciences College of Osteopathic Medicine, Kansas City, MO in 1970. He completed his residency at Menninger Clinic, Topeka, KS. Dr. Curran specializes in psychiatry.

Jean Diemer, M.D., 14 Doctors Park, Cape Girardeau, MO 63701, has become a member of the Cape Girardeau County Medical Society. Dr. Diemer received her baccalaureate degree from Southern Illinois University, Carbondale, IL and obtained her medical degree from the

University of Illinois at Peoria in 1988. She completed her residency at St. Francis Medical Center, Peoria, IL. Dr. Diemer specializes in pediatrics.

Thomas S. Diemer, M.D., 14 Doctors Park, Cape Girardeau, MO 63701, has become a member of the Cape Girardeau County Medical Society. Dr. Diemer received his baccalaureate degree from the University of Illinois-Urbana and obtained his medical degree from the University of Illinois at Peoria in 1988. He completed his residency at St. Francis Medical Center, Peoria, IL. Dr. Diemer specializes in internal medicine.

Sandy R. Dillard, M.D., 2617 Cunningham, Suite B, Joplin, MO 64804, has become a member of the Jasper County Medical Society. Dr. Dillard received his baccalaureate degree from Kansas State University and obtained his medical degree from the University of Kansas School of Medicine, Lawrence-Kansas City in 1987. He completed his residency at the University of Kansas School of Medicine-Wichita Affiliate Hospital. Dr. Dillard specializes in anesthesiology.

David B. Dunn, M.D., 118 Westwoods Dr., Liberty, MO 64068, has become a member of the Clay-Platte County Medical Society. Dr. Dunn received his baccalaureate degree from Texas A & M University, College Station and obtained his medical degree from the University of Texas Health Science Center, San Antonio in 1985. He completed his residency at Scott & White Memorial Hospital, Temple, TX. Dr. Dunn specializes in general vascular surgery.

Melanie A. Dunn, M.D., 17500 Medical Center Parkway, Suite 3, Independence, MO 64057, has become a member of the Missouri State Medical Association. Dr. Dunn received her baccalaureate degree from Texas A & M University, College Station and obtained her medical degree from Texas A & M the University College of Medicine in 1987. She completed her residency at Scott & White Memorial Hospital, Temple, TX. Dr. Dunn specializes in obstetrics and gynecology.

Shelley J. Edwards, M.D., 2929 Baltimore, Suite 415, Kansas City, MO 65108, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Edwards received her baccalaureate degree from Southern Illinois University at Edwardsville and obtained her medical degree from the University of Kansas School of Medicine, Kansas City in 1988. She completed her residency at the University of Kansas, Kansas City. Dr. Edwards specializes in internal medicine.

James L. Harwell, M.D., 4502 N. Fallwood Ct., Columbia, MO 65203, has become a member of the Boone County Medical Society. Dr. Harwell received his baccalaureate degree from the University of Missouri-Columbia and obtained his medical degree from the University of Missouri-Columbia School of Medicine in 1984. He completed his residency at the University of Missouri-Columbia Hospitals & Clinics. Dr. Harwell specializes in internal medicine.

John M. Hoja, M.D., 1901 Sunset, Poplar Bluff, MO 63901, has become a member of the Butler-Wayne-Ripley-Carter County Medical Society. Dr. Hoja received his baccalaureate degree from the University of Missouri-Kansas City and obtained his medical degree from the University of Missouri School of Medicine-Kansas City in 1978. He completed his residency at Stamford Hospital, Stamford, CT. Dr. Hoja specializes in obstetrics and gynecology.

Jonathon H. Johnson, M.D., R.R. 6, Box 711, Poplar Bluff, MO 63901, has become a member of the Butler-Wayne-Ripley-Carter County Medical Society. Dr. Johnson received his baccalaureate degree from the University of Missouri-Columbia and obtained his medical degree from Eastern Virginia Medical School-Norfolk in 1984. He completed his residency at Baptist Memorial Hospital, Memphis, TN. Dr. Johnson specializes in pathology.

Rebecca S. Johnson, M.D., 6420 Prospect, Suite T107, Kansas City, MO 64132, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Johnson received her baccalaureate degree from Iowa State University, Ames and obtained her medical degree from the University of Iowa College of Medicine, Iowa City in 1980. She completed her residency at the University of Michigan Affiliate Hospitals, Ann Arbor. Dr. Johnson specializes in obstetrics and gynecology.

Ronald J. Lakusiewicz, M.D., 2509 Lucy Lee Parkway, Poplar Bluff, MO 63901, has become a member of the Butler-Wayne-Ripley-Carter County Medical Society. Dr. Lakusiewicz received his baccalaureate degree from Seton Hall University, Newark, NJ and obtained his medical degree from the University of New Jersey Medical and Dental School, Newark, NJ in 1970. He completed his residency at Southeastern Michigan Plastic Surgical Program, Detroit, MI. Dr. Lakusiewicz specializes in plastic surgery.

David M. Lewis, M.D., 1118 N. Main, Sikeston, MO 63801, has become a member of the SEMO Medical Society. Dr. Lewis received his baccalaureate degree from Brigham Young University, Provo, UT and obtained his medical degree from the University of Utah School of Medicine, Salt Lake City in 1984. He completed his residency at the University of Louisville. Dr. Lewis specializes in otolaryngology.

Roy D. Libel, M.D., 3231 South National, Springfield, MO 65807, has become a member of the Greene County Medical Society. Dr. Libel received his baccalaureate degree from the University of Kansas and obtained his medical degree from the University of Kansas School of Medicine, Lawrence-Kansas City in 1986. He completed his residency at the University of Kansas. Dr. Libel specializes in gastroenterology.

Patrick T. Little, D.O., 909 E. Reed, Hayti, MO 63851, has become a member of the Pemiscot County Medical Society. Dr. Little obtained his medical degree from the University of Health Sciences College of Osteopathic Medicine, Kansas City in 1974. Dr. Little specializes in obstetrics and gynecology.

Vickie L. Massey, M.D., 6400 Prospect, Suite 240, Kansas City, MO 64132, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Massey received her baccalaureate degree from the University of Missouri-Kansas City and obtained her medical degree from the University of Missouri-Kansas City School of Medicine in 1985. She completed her residency at Memorial Sloan Kettering Cancer Center, New York, New York. Dr. Massey specializes in radiation therapy.

Mark Melew, M.D., 14854 Grantley, Chesterfield, MO 63017, has become a member of the Missouri State Medical Association. Dr. Melew received his baccalaureate degree from the University of Denver, Denver, CO and obtained his medical degree from Washington University School of Medicine, St. Louis, MO in 1986. He completed his residency at the University of Miami, Jackson Memorial Medical Center. Dr. Melew specializes in orthopedics.

John R. Patty, D.O., 1901 Sunset Drive, Poplar Bluff, MO 63901, has become a member of the Butler-Wayne-Ripley-Carter County Medical Society. Dr. Patty received his baccalaureate degree from Southeast Missouri State University, Cape Girardeau and obtained his medical degree from Kirksville College of Osteopathic Medicine, Kirksville, MO in 1986. He completed his residency at Southern Illinois University, Springfield, IL. Dr. Patty specializes in obstetrics and gynecology.

José A. Remo, Jr., M.D., 233-C West Springfield Rd., Sullivan, MO 63080, has become a member of the Missouri State Medical Association. Dr. Remo received his baccalaureate degree from the University of the Philippines, Quezon City and obtained his medical degree from Perpetual Heal College of Medicine, Binan, Laguna, Philippines in 1982. He completed his residency at the University of Missouri-Columbia Hospitals & Clinics. Dr. Remo specializes in internal medicine.

Debra L. Royce, M.D., 1407 Miner St., Sarcoxie, MO 64862, has become a member of the Jasper County Medical Society. Dr. Royce received her baccalaureate degree from Drury College, Springfield, MO and obtained her medical degree from St. Louis University School of Medicine, St. Louis, MO in 1988. She completed her residency at Truman Medical Center-East, Kansas City, MO. Dr. Royce specializes in family practice.

Joni C. Scott, M.D., 1965 S. Fremont, Springfield, MO 65804, has become a member of the Greene County Medical Society. Dr. Scott received her baccalaureate degree from Milligan College and obtained her medical degree from the University of Oklahoma College of Medicine, Oklahoma City in 1984. She completed her residency at the University of Oklahoma Health Sciences Center. Dr. Scott specializes in general surgery.

Charles F. Sherrod, M.D., 1531 W. 32nd, Suite 102, Joplin, MO 64804, has become a member of the Jasper County Medical Society. Dr. Sherrod received his baccalaureate degree from Louisiana Tech University and obtained his medical degree from Louisiana State University Medical Center, Shreveport School of Medicine in 1987. He completed his residency at Louisiana State University Medical Center. Dr. Sherrod specializes in ophthalmology.

Ricky A. Smith, M.D., 1012 N. Main, Sikeston, MO 63801, has become a member of the SEMO Medical Society. Dr. Smith received his baccalaureate degree from Memphis State University, Memphis, TN and obtained his medical degree from the University of Tennessee Center for Health Sciences, Memphis in 1983. He completed his residency at the University of Tennessee, Memphis. Dr. Smith specializes in urology.

Michael E. Somers, M.D., 2700 Clay Edwards Drive, North Kansas City, MO 64116, has become a member of the Clay-Platte County Medical Society. Dr. Somers received his baccalaureate degree from Brown University, Providence, RI and obtained his medical degree from Brown University of Biological-Medical Sciences, Providence, RI in 1894. He completed his residency at Yale Eye Center, New Haven, CT. Dr. Somers specializes in ophthalmology.

Robert F. Stringer, D.O., 530 E. 34th St., Suite 203, Joplin, MO 64804, has become a member of the Jasper County Medical Society. Dr. Stringer received his baccalaureate degree from Pittsburg State University, Pittsburg, KS and obtained his medical degree from the University of Health Sciences College of Osteopathic Medicine, Kansas City, MO in 1986. He completed his residency at Tulsa Regional Medical Center, Tulsa, OK. Dr. Stringer specializes in orthopedic surgery and medicine.

Jim Suthoff, D.O., 2617 Cunningham, Suite B, Joplin, MO 64804, has become a member of the Jasper County Medical Society. Dr. Suthoff received his baccalaureate degree from the University of Missouri and obtained his medical degree from Kirksville College of Osteopathic Medicine, Kirksville, MO in 1985. He completed his residency at Botsford General Hospital, Farmington Hills, MI. Dr. Suthoff specializes in anesthesiology.

Richard P. Valuck, Jr., M.D., 109 Virginia, Hannibal, MO 63401, has become a member of the Northeast Medical Society. Dr. Valuck received his baccalaureate degree from the University of Missouri-Columbia and obtained his medical degree from the University of Missouri School of Medicine-Columbia in 1985. He completed his residency at the University of Cincinnati. Dr. Valuck specializes in cardiology.

Corrections:

In the January 1992 Issue of *Missouri Medicine* (New Members Section), Drs. **William C. Parks** and **Larry A. Scroggins** should have been listed as specializing in Emergency Medicine; not Family Practice. In the December 1991 Issue, Dr. **Michael Critchlow** should have been listed as specializing in Allergy/Clinical Immunology; not internal medicine.

RESIDENT SECTION

Sudhir S. J. Batchu, M.D. neurology
Carlos R. Berrios, M.D. orthopedics
Kevin J. Black, M.D. psychiatry
Lesli A. Bourassa, M.D. general surgery
Tonya T. Cooley, D.O. internal medicine
Rachel L. Dotson, M.D. internal medicine
Rebecca A. Haefner, M.D. anesthesiology
W. Steven Hughes, M.D. general surgery
Paul W. Humphrey, M.D. general surgery
Christopher M. Ottinger, M.D. family practice

STUDENT SECTION

St. Louis University
Virginia A. Thompson

University of Missouri-Kansas City
Christian S. Hinrichs
Mary E. Keller
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Washington University
Mike C. Kim
Nichol M. Trump

One Year Ago

Victory in the Gulf War

America held its breath last February as this nation's men and women made final preparations to fight a war. Across the sandy plains, only a few miles away, battle tested troops waited for their advance. Iraq's soldiers were pledged to defend their homeland and way of life against the influences of Western civilization, and they were led by Saddam Hussein, a ruler that President Bush compared to Hitler.

The war had begun January 17, 1991 when allied aircraft attacked Hussein's forces in Iraq and Kuwait with what grew to be a 39-day long, textbook perfect, aerial bombardment. Once the Air Force finished, however, ground grunts would have to step on to the battleground and, just as in every war that has ever been fought, subdue the enemy.

The issues that pulled the United States into this war: preserving access to oil resources, containing a military leader who was developing massive nuclear, biological and chemical threat capabilities, and responding to a call to help the people of Kuwait, galvanized America's support and attention.

And, this was not to be another Viet Nam-type stalemate. Flags flew across the land, demonstrating a popular support for American troops that had not been seen since World War II.

Thankfully, victory was swift. One year later, people remember with gratitude and pride America's commitment to decisive action.

Photo by Eugene W.J. Pearce, M.D.

At the peak of the Gulf War, an American flag and yellow ribbon, above, adorned a statue of Harry S. Truman that stands in the foyer of the Truman Medical Center in Kansas City.

Medical School News

Washington University

The School of Medicine at Washington University has restructured it tuition policy to freeze costs throughout the four years of medical school for students enrolling in the 1992-93 academic year.

For new students entering the school in August 1992, annual tuition will be $19,800 and will be frozen at that amount for all four years of their medical education. For currently enrolled students, tuition will be increased five percent, from $15,900 to $16,700.

The indebtedness of Washington University medical students is below the national average. Large educational debts have been associated with young physicians' tendencies to select higher-paying specialties and to avoid careers in primary care and academic medicine.

Diabetics, age 18 or older, who take insulin and are on immunosuppressive medications or are being seen by a nephrologist for potential kidney failure are being asked to volunteer for an experimental islet transplant procedure at Washington University Medical Center. The study is designed to reduce or possibly eliminate insulin dependence. In the transplant process, islets are collected from donor pancreas tissue, purified and injected into the recipient. If the transplant is successful, the transplanted islet cells take over glucose regulation by secreting appropriate levels of insulin, thus reducing or erasing the patient's need for insulin injections. Those individuals accepted for the study will receive their transplants and clinical care at Barnes Hospital.

Researchers at Washington University are testing an experimental drug treatment to determine if it will halt shingles sooner than current medication and if it can prevent the long-term pain often associated with shingles. Shingles is the result of reactivation of the chickenpox virus.

UNIVERSITY OF MISSOURI-COLUMBIA

The Robert Wood Johnson Foundation of Princeton, J.J., has selected the MU School of Medicine's department of family and community medicine to direct its new $32.7 million national Initiative to Increase the Supply of Generalist Physicians.

The foundation, established as a national philanthropy in 1972, has awarded more than a billion dollars in grant funds with the goal of bringing better health care to the American people. The new seven-year initiative is designed to address a nationwide shortage of primary health-care providers by encouraging medical students to choose careers as family practitioners, general internists and general pediatricians.

"How physicians are distributed according to specialty has important implications for the kind of medical care given, who gets it and where, and how much it costs," said **Steven A. Schroeder, M.D.,** RWJF president. "A system with excess specialists in inefficient and expensive, and creates access barriers.

"Inadequate access exists in both inner city and rural areas. The result is either inefficient and expensive alternatives such as the use of emergency rooms for non-urgent problems or doing without care," he explained.

The initiative will be directed by **Jack M. Colwill, M.D.,** chairman of the MU department of family and community medicine and a nationally respected authority on primary care and medical education. the deputy directors will be two of his department's faculty members, Professor **Robert L. Blake Jr., M.D.,** and Professor Emeritus **Gerald T. Perkoff, M.D.**

"The shortage of primary-care physicians is the single most important issue in physician manpower today," Colwill said. "While the nation's medical schools in the past 25 years have more than doubled their output, the preponderance of graduates is progressively seeking subspecialties. Consequently, only about one medical graduate in five is planning a generalist career."

The competitive program will be carried out in two stages. At least two-thirds o the nation's medical schools are expected to meet the May deadline to apply for 18 available one-year planning grants of up to $150,000 each. After the year is up, as many as 12 schools with promising plans will receive operational grants of up to $2.5 million each to implement their programs over the next six years.
maintenance organizations, hospitals and community health centers.

James Cassidy, M.D., professor of child health, recently received the Earl Brewer Award for health Care Professionals from the American Juvenile Arthritis Organization. The award, now in its third year, is given annually to a health-care professional for activities addressing the issue of arthritis in children locally and nationally. One award each is presented to a patient, a lay individual and a health professional. Cassidy, a founder of the organization in 1980, was active on its executive board for six years and wa instrumental in initiating the organization's regional meetings.

Bernard G. Ewigman, M.D., associate professor of family and community medicine, has been selected to serve on the planning committee for the national Primary Care Conference scheduled for march 1992. It is jointly sponsored by the Health Care Financing Administration and the Public Health Service.

In addition, Ewigman recently was appointed chairman of the Research Committee for the Society of Teachers of Family Medicine.

Ramesh Khanna, M.D., professor of nephrology, spoke on a variety of topics dealing with peritoneal dialysis during recent trips to Campinas, Brazil; Quito, Ecuador; and Taejon, Korea. The groups he addressed included the Latin American Congress on Peritoneal Dialysis and the Koran Society of Nephrology.

Robert McMurray, M.D., MU clinical instructor of immunology and rheumatology nd a staff physician at Harry S. Truman Memorial Veteran's Hospital, is the 1991 recipient of the Trainee Award for the Midwest section of the American Federation for Clinical Research. The award, given to five individuals this year, is based on the quality of work performed, the primary role of the trainee in the investigation and the support of the mentor. McMurray's research adviser was **Sara E. Walker, M.D.**, MU professor of immunology and rheumatology. His research described the hormone prolactin's effect on systemic lupus erythematosus disease activity in animal models.

Marian A. Minor, Ph.D., assistant professor of physical therapy and a researcher with the Missouri Arthritis Rehabilitation Research Center has been elected program chairperson for the Arthritis Health Professions Association's 27th annual National Scientific Meeting in 1992.

Karl T. Weber, M.D., professor and chairman of internal medicine, recently gave a presentation to the 32nd Japanese College of Angiology Congress Satellite Symposium in Tokyo. He spoke on "Prevention and Regression of Left Ventricular Hypertrophy and Myocardial Fibrosis by Spironolactone in Various Hypertension Models."

UNIVERSITY OF MISSOURI-KANSAS CITY

E. Grey Dimond, M.D., founder and architect of the UMKC six-year medical school program, has published his autobiography, *Take Wing!* The 400-page book, including 16 pages of photos, relates Dr. Dimond's experiences as a physician-teacher, cardiologist-consultant, educator-author, administrator-leader and experienced world traveler. It also recalls many activities associated with the founding and early growth of UMKC's medical school.

Dr. Dimond has donated the *Take Wing!* manuscript and all proceeds from the sale of his book to a special UMKC School of Medicine Alumni fund.

Several versions of limited first-edition copies are available. For more information, contact the Office of Alumni and Development, UMKC School of Medicine, 2411 Holmes, Kansas City, MO 64108-2792, 816-235-5281.

Dean **James J. Mongan, M.D.**, was featured in a cover story, "CEOs Confront the Ethics of Resource Management: Three Nationally Known Hospital CEOs Respond to a Situational Scenario," in the Jan. 5, 1992, issue of *Hospitals*, a magazine for health care executives. Dr. Mongan, executive director of Truman Medical Center, was one of three hospital chiefs who outlined how he would allocate limited capital funds given equipment needs in three different hospital departments.

Pfizer Laboratories recently awarded the cardiology section of the Department of Medicine a $5,000 grant for the Pfizer Laboratories Visiting Professorship in Cardiovascular Disease. Internationally renowned Cardiologist Dr. Shahbudin Rahintoola will be the visiting professor March 11-13.

William L. Mundy, M.D., clinical professor of medicine, presented a workshop on "Transactional Analysis and Gestalt Techniques for Working with Psychosomatic Disease" at the Third National Conference of the national Institute for the Clinical Application of Behavioral Medicine December 4-7, 1991, in Orlando, Florida.

Steve O. Stephenson, Kevin C. Wright, M.D., instructor of radiology, and **Christopher J. Meoli, D.O.**, assistant professor of radiology, presented a poster and scientific exhibit on "Realistic 3-D Visualization of Joint Surfaces and Soft Tissue from CT Data" at the annual meeting of the Radiologic Society of North America December 2-6, 1991, in Chicago, Illinois.

ST. LOUIS UNIVERSITY MEDICAL CENTER

A new study by a St. Louis University researcher indicates that electroconvulsive therapy (ECT) could be one of the safest and most effective ways of treating severe depression in the elderly. Electroconvulsive therapy is a treatment alternative for the elderly who get no relief from standard therapy or medication, or those who are sensitive to medication. Researchers say ECT should be considered as a first line of treatment in patients who are suicidal or starving themselves because it is important to treat these patients quickly and effectively.

Over a two year period, researchers studied 57 elderly patients 65-years-of-age and older. Each patient was evaluated before treatment to determine whether they were physically fit. Although many of the patients had pre-existing medical problems and even heart conditions, they qualified for the treatment.

In none of the cases did the therapy exacerbate physical problems. In addition, a majority of the patients between 65 and 80, and all of the patients 80 years and older experienced relief from their depression. They showed either moderate or significant improvement in their symptoms and functioning. There were two cases of prolonged confusion, which cleared in 48 hours. There were no cases of prolonged memory impairment and there was one fall but with no major consequences.

"What this shows is that age should not be a barrier to this effective treatment," said **Jothika Manepalli, M.D.**, director, late life mood disorders program, department of psychiatry and human behavior, St. Louis University School of Medicine. "Doctors should not be afraid to aggressively treat severe depression in the elderly."

Doctors at St. Louis University School of Medicine are testing a new drug that might heal the painful and dangerous ulcers and prevent the loss of limbs affiliated with uncontrolled diabetes.

The drug is known as the acidic fibroblast growth factor, a topical solution that stimulates the growth of skin when spread on a damaged area.

"The treatment of non-healing ulcers is so undeveloped that any contribution would be major," said Arshag D. Mooradian, M.D., professor of Internal medicine in the division of endocrinology and coordinator of the study. St. Louis University School of Medicine is one of only four facilities in the country testing the drug.

Researchers at St. Louis University School of Medicine are testing an investigational nasal steroid spray designed to control non-allergic rhinitis.

"Non-allergic rhinitis is very common, affecting about 20 percent of the population," said Howard M. Druce, M.D., associate professor of internal medicine in the division of allergy and immunology and assistant professor of otolaryngology, St. Louis University School of Medicine. "The problem is especially prevalent in areas with high humidity such as St. Louis. Most people use over-the-counter decongestants to relieve their symptoms. The nasal spray, however, may prove to be a better control."

Preliminary studies in Europe indicate the spray is effective in controlling the problem.

Marc J. Shapiro, M.D., associate professor os surgery and director of the division of trauma services, St. Louis University School of Medicine, has received two grants totaling $110,400 to study ways to reduce trauma and surgical infections.

A one-year, $80,400 grant from Synergen/Pharmaco Dynamics Research, Inc. will be used for an international study evaluating the "Safety and Efficacy of Human Recombinant Interleukin-1 Receptor Antagonist (IL-1ra) in the Treatment of Sepsis Syndrome." Researchers are using the protein, IL-1ra, to block the body's natural reaction to surgical and trauma-related infections, such as overwhelming pneumonia and abdominal infections. This is the first world-wide clinical study to examine how to eliminate interleukin's effect on the body.

A six-month, $30,000 grant from Merck Sharp and Dohme is being used to study "Primaxin 1.V. in the Treatment of Serious Intra-Abdominal infections." This is one of an ongoing series of investigations by the division of trauma services looking into the effectiveness of various antibiotics in preventing infections in penetrating wounds such as gun shot or stab wounds to the abdomen. Researchers are currently evaluating results of the effectiveness of one antibiotic called Primaxin.

Frank V. Aguirre, M.D., assistant professor of cardiology in the department of internal medicine, St. Louis University School of Medicine, has received a $70,000 grant from Genentech, Inc. to coordinate the "t-PA-Heparin Duration Study." The multicenter study will try to determine how long doctors should treat patients with Heparin, a blood thinner.

"We're trying to determine whether one day of Heparin is just as good as three days," Dr. Aguirre said. "If one day is enough to keep the arteries open, we cut down on possible bleeding complications and we can also cut down on medication costs to the patient."

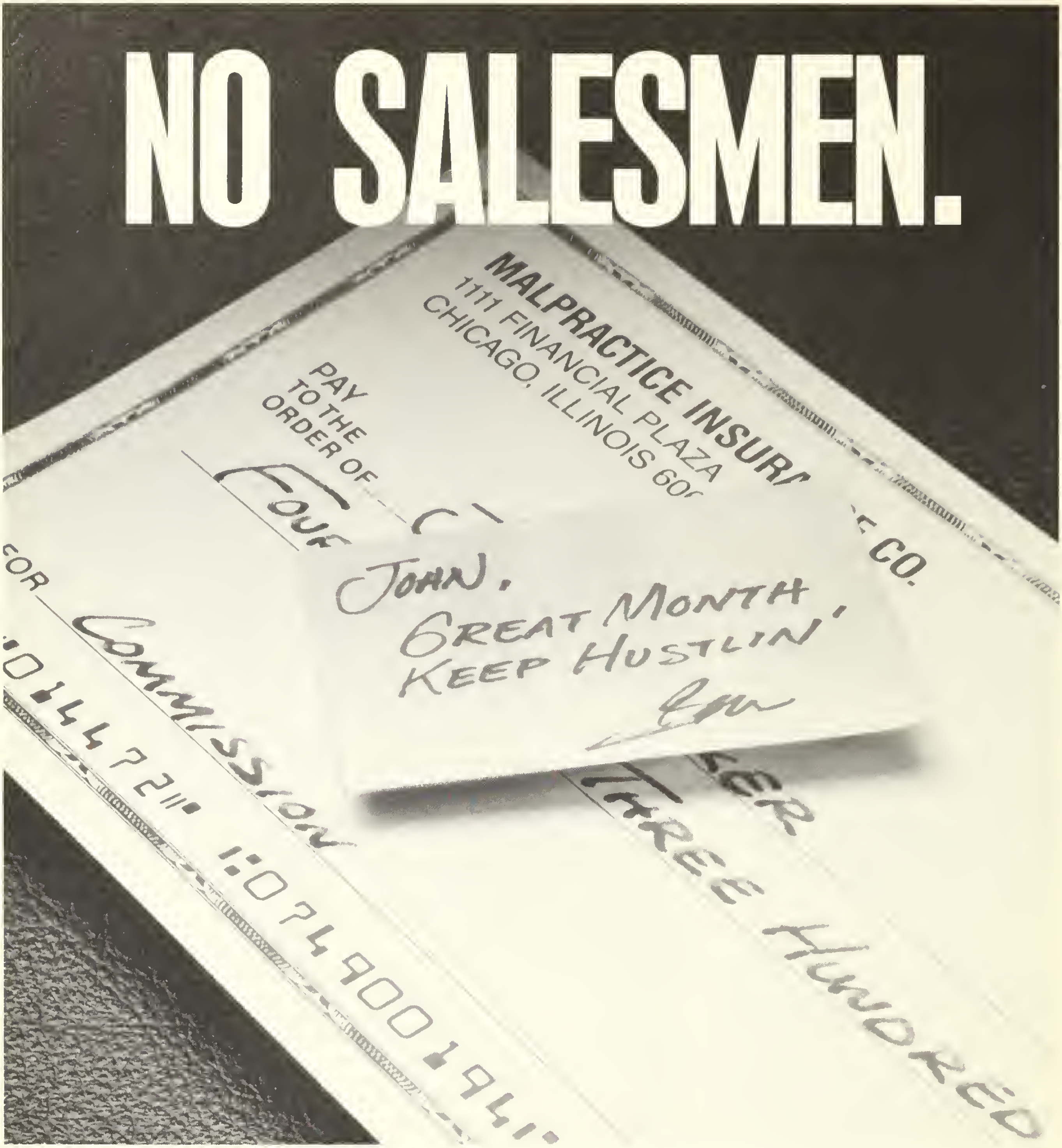
Dr. Aguirre will examine the clinical outcomes and angiograms of 450 patients throughout the Midwest who will participate in the drug study. The patients will be treated with Heparin for varying lengths of time and Dr. Aguirre will look for signs of reclotting, bleeding complications and recurring heart attacks.

Researchers at St. Louis University School of Medicine have received a $64,500 grant from Parke-Davis, the Pharmaceutical Research Division of the Warner Lambert Company, to test a new form of the antiseizure drug phenytoin. Phenytoin is given to epilepsy patients to control seizures and to patients with head injuries who are prone to seizures.

Currently, phenytoin can be delivered in only two ways: orally or intravenously. The difficulties with delivering the medication by mouth is that a patient may be unconscious or unable to swallow. Administering the drug intravenously causes irritation to the veins. Also, if the medication is given too quickly, it can cause cardiac arrythmia or heart block.

Researchers are working with fosphenytoin, a new form of phenytoin that can be injected into muscles of the buttocks. The multicenter study is designed to test the safety and tolerance of delivering the drug intramuscularly. Preliminary results indicate that no serious dermatologic or muscular reactions have occurred with fosphenytoin administration.

Kenneth R. Smith Jr., M.D., professor of surgery, director of the neurosurgery division and coordinator of the study said, "Patients who have poor venous access or who cannot swallow will now be able to receive fosphenytoin while keeping the serum blood levels in an acceptable range."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FEBRUARY

3 - 6 **Practical Anatomy & Surgical Technique Workshop Temporal Bone Surgical Dissection** - Hand-on workshop. Location: St. Louis Metropolitan Medical Society Building. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

7 **Otology Update** - Location: St. John's Mercy Medical Center. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

7 **Kawsaki Disease** - Location: Clopton Auditorium. Contact: Department of Pediatrics, Washington University School of Medicine and St. Louis Children's Hosptial

7 **Interventional Radiology** - Location: ResearchMedical Center. Contact: The Ed Bixby Institute for Postgraduate Medical Education, 2316 East Meyer Boulevard, Kansas City, Missouri 64132. Missouri: (800) 892-5771, ext. 4306. Bordering states: (800) 821-5140, ext. 4306.

7 - 8 **Midwinter Laser Course & Fluorescein Angiography Workshop** - Location: Hyatt Regency Hotel, St. Louis, MO. Contact Leigh Dundon, Suite 17413, East Pavilion, One Barnes Hospital Plaza, St. Louis, MO 63110. 314/367-1181.

10 **Evaluation of Myocardial Viability** - Location: Mallinckrodt Institute of Radiology, St. Louis, Mo. Contact: R. Gilbert Jost, M.D., chief of Diagnostic Radiology, Mallinckrodt Institute, 510 South Kingshighway, St. Louis, MO 63110. 314/362-7130.

11 - 12 **Introduction to Doppler Echocardiography** - Location:Saint Louis University Medical Center. Contact: CME Office, SLU, 800/553-2712 or 577-8167.

14 **Drug Abuse and High Risk Behavior in St. Louis Population** - Location: Department of Pediatrics, Washington University School of Medicine and St. Louis Children's Hosptial.

19 - 21 **Twelfth Annual Conference on Peritoneal Dialysis** - Location: Seattle Convention Center, Seattle, Washington. Contact Barry Kling, UMC, MA215 Health Sciences Center. 314/882-4105.

28 **Interferon and other Therapy for Chronic Viral Hepatitis** - Location: Department of pedicatrics, Washington University School of Medicine and St. Louis Children's Hosptial.

28 **Lyme Disease** - Location: Bartle Hall, Room 214 North Side of 13th & Central, Kansas City, MO. Contact: The Ed Bixby Institute for Postgraduate Medical Education, Research Medical Center 2316 East Meyer Boulevard, Kansas City, MO 64132.

MARCH

3 - 4 **Transesophageal Echocardiography Course** - Location: Saint Louis University Medical Center. Contact: CME Office, SLU, 1402 S. Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 **Host-Pathogen Interactions in Microbial Pathogensis** - Location: 3rd floor Auditorium, Children's Hospital. Contact: Department of Pediatrics, Washington University School of Medicine.

6 **Controversies in the Treatment of Early Breast Cancer and in the Treatment of Gynecological Cancer** - Location: The Ritz-Carlton Hotel, St. Louis, MO. Contact: St. Louis University Medical Center CME Office at 1-800-553-2712 or 314-577-8167.

6 **Current Perspectives in Cancer Therapy, The Multi-modal Approach** - Location: The Ritz-Carlton Hotel, St. Louis, MO. Contact: CME Office, SLU , 1402 South Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 - 7 **Advanced Trauma Life Support Provider** - Location: Margaret McCormick Doisy Learning Resources Center. Contact: CME Office, SLU, 1402 South Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 - 8 **Family Practice Update** - Location: UMC School of Medicine, Columbia, MO. Contact: Barry Kling, CME Office, MA215 Health Sciences Center. 314/882-4105.

7 **Flexible Sigmoidoscopy** - Location: DePaul Health Center, St. Louis Seton Auditorium and Classroom 3. Contact: Sheri Leppanen, Depaul Health Center, (314) 344-6000, ext. 2339.

9 **CT of the Lung Parenchyma** - Location: Mallinckrodt Institute of Radiology. Contact: R. Gilbert Jost, M.D., Mallinckrodt Institute of Radiology, 510 S. Kingshighway Boulevard, St. Louis, MO 63110. 314/362-7130.

13 **Orbit and Eyelid Surgery** - Location: Margaret McCormick Doisy Learning Resources Center, Saint Louis University. Contact: CME Office, SLU, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

15 - 20 **Difficult Situations in Clinical Practice (14th Annual Winter Psychiatry Conference)**- LocationPark City, Utah. Contact: The Menninger Clinic, Box 829, Topeka, KS 66601-0829.

27 **Tenth Annual Geriatric Symposium** - Location: Breckenridge Frontenac, St. Louis, MO. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

APRIL

2 - 3 **2-3 Annual Obstetrics & Gynecology Symposium** - Location: Washington University Medical Center, St. Louis, MO. Contact: CME Office, Box 8063, 660 South Euclid Avenue, St. Louis, MO 63110-1093

4 **Liver Transplantation for the Primary Care Physician**- Location: Margaret McCormick Doisy Learning Resources Center, St. Louis University. Contact: CME Office, SLU, 1412 South Grand blvd., St. Louis, MO 63104. 314/577-8167.

13 **City-Wide Radiology Conference** - Location: Mallinckrodt Institute of Radiology, Washington University Medical Center. Contact: R. Gilbert Jost, M.D., 510 South Kingshighway Blvd. St. Louis, MO 63110. 314/362-7130.

MAY

7 - 10 **Emergency Department Legal Environment** - Location: St. Petersburg Beach, Florida. Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

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Manuscripts should be typewritten, double-spaced, and the original with one copy submitted. Retain another copy for proofreading. Please number the pages of your manuscript. Manuscripts are not returned. Hospital and University appointments of the author(s) should accompany the manuscript. A synopsis-abstract of no more than 75 words should accompany the manuscript. Manuscripts may be submitted on 5¼ or 3½ floppy disks using WordPerfect 5.1 or below.

References

References should be cited in numerical order in the text and arranged at the end of the article in the same order in which they are cited. The reference should give name of author, title of article, name of periodical, volume number, initial page number and year. Authors are responsible for reference accuracy.

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Editorials should be written in clear, concise language. Length should be about two or three pages typed with double spacing. Letters will be published at the discretion of the editor, managing editor, and Editorial Board.

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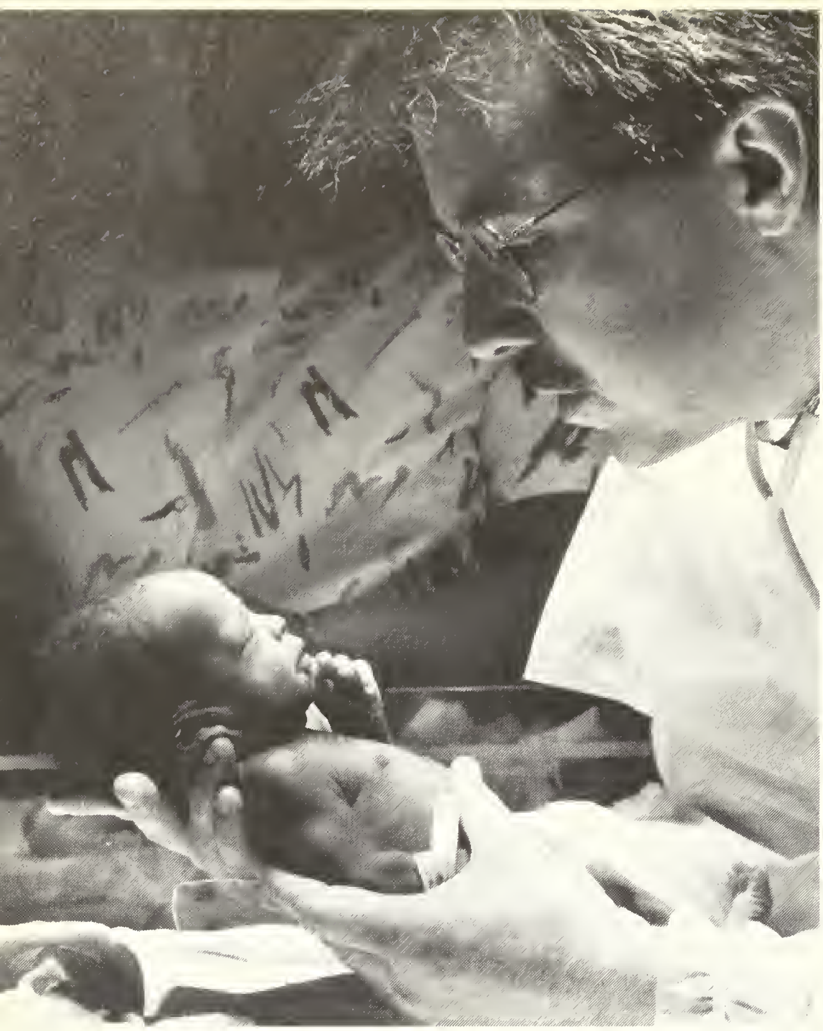

"As doctors, if we do the right thing at the right time, we can make a difference."

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AIR FORCE

Letters to the Editor

Dear Dr. Sessions:

Enclosed is an article simulating Dr. Dorothy D. Reister's article in *Missouri Medicine* October issue offering suggestions as to how a doctor may be still more helpful and appreciative in his community.

I read and admired the article written by our honorable president, Dorothy D. Reister, printed on the President's Page of *Missouri Medicine*, October 1991, Vol. 88, No. 10, page 687, whereby doctors may be associated with various phases of medical activities. One other suggestion for doctors of Missouri and elsewhere is to offer a scholarship to a high school senior graduate in their immediate community where they practice medicine. To endorse and comply to this suggestion, I have through a period of years given fifty-one scholarships to Central Missouri State University, to a senior graduate of Grandview, Belton, Savannah and Rich Hill Missouri High Schools.

Sam D. Hoeper Sr., M.D.
Grandview, MO

Dear Dr. Sessions:

The editorial on Quality of Care in medicine (*Missouri Medicine:* Vol. 88, No. 12, page 799-800) in the state medical journal fails to recognize that the issue of quality is more of a reality today than ever before. To say that quality in medicine cannot be determined is like saying that religion is an elixir of the weak.

Quality is a term used frequently and loosely, in the past, and today to discriminate against a rival in medical practice. Quality is still determined on the basis of the place of education, his alma mater and so forth. Glory of a stable lies in the downfall of his neighbors. However, we all forget that quality is determined by the ultimate consumer.

Quality is defined as when a product or service meets and exceeds the consumer's expectation. If the physician community asserts that quality cannot be determined in medicine, then we are like a ladle in soup, which neither imparts any taste to the soup, nor is benefited by it. It is much better to participate in the process of developing the quality, and in the process be destroyed if that is the fate, than to be destroyed by an outsider.

Ashokkumar B. Patel, M.D., M.P.H.
St. Louis, MO

Physicians's Charity Care Program Update

Charity care, as defined for this program, includes hands-on medical care rendered in any practice setting, to indigient patients (those without funds), Medicaid patients, or both. Professional courtesy, bad debt cases, and Medicare patients are not included. The program is voluntary and is open to members and non-members of the MSMA and AMA.

The Missouri State Medical Association recognizes physicians who give 50 or more hours of hands-on charity care.

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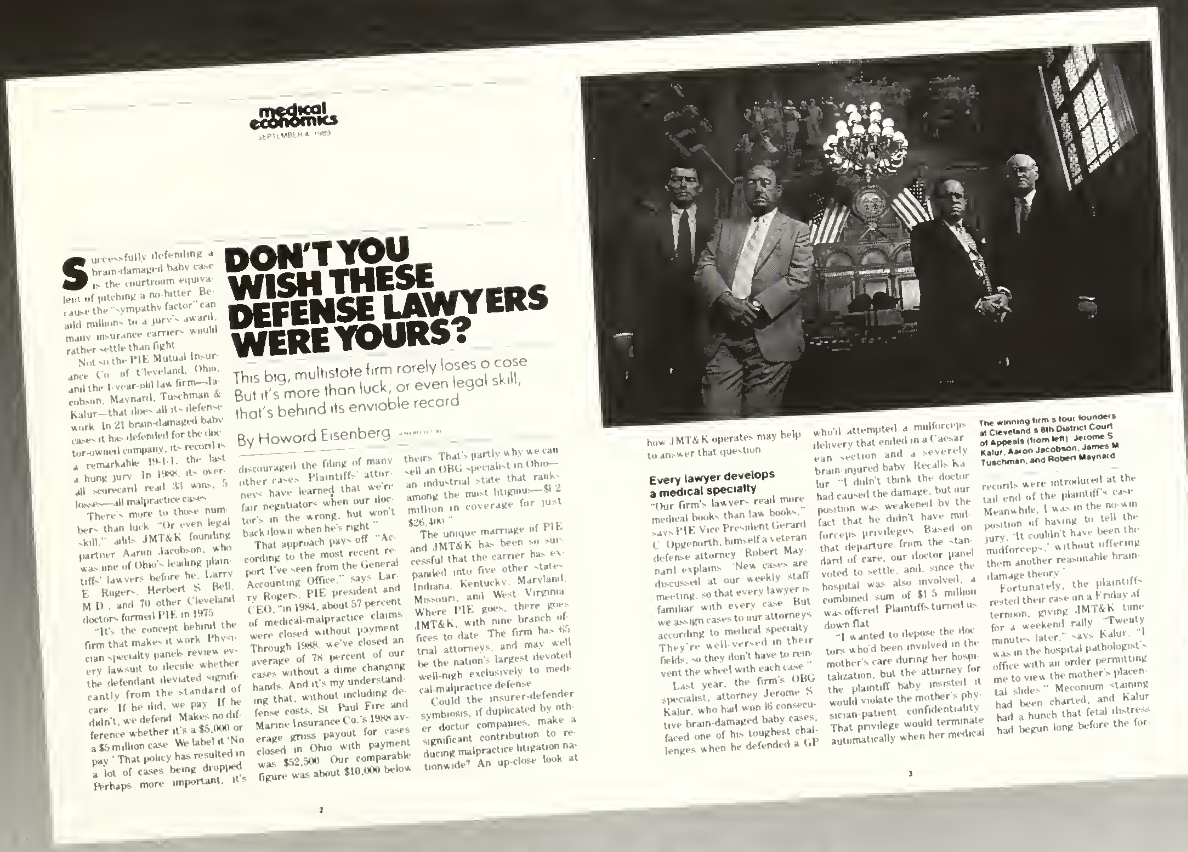

medical economics
SEPTEMBER 4, 1989

DON'T YOU WISH THESE DEFENSE LAWYERS WERE YOURS?

This big, multistote firm rorely loses o cose. But it's more than luck, or even legal skill, that's behind its enviable record

By Howard Eisenberg

Successfully defending a brain-damaged baby case is the courtroom equivalent of pitching a no-hitter. Because the "sympathy factor" can add millions to a jury's award, many insurance carriers would rather settle than fight.

Not so the PIE Mutual Insurance Co. of Cleveland, Ohio, and the 14-year-old law firm—Jacobson, Maynard, Tuschman & Kalur—that does all its defense work. In 21 brain-damaged baby cases it has defended for the doctor-owned company, its record is a remarkable 19-1-1, the last a hung jury. In 1988, its overall scorecard read 33 wins, 5 losses—all malpractice cases.

There's more to those numbers than luck. "Or even legal skill," adds JMT&K founding partner Aaron Jacobson, who was one of Ohio's leading plaintiffs' lawyers before he, Larry E. Rogers, Herbert S. Bell, M.D., and 70 other Cleveland doctors formed PIE in 1975.

"It's the concept behind the firm that makes it work. Physician specialty panels review every lawsuit to decide whether the defendant deviated significantly from the standard of care. If he did, we pay. If he didn't, we defend. Makes no difference whether it's a $5,000 or a $5 million case. We label it 'No pay.' That policy has resulted in a lot of cases being dropped. Perhaps more important, it's discouraged the filing of many other cases. Plaintiffs' attorneys have learned that we're fair negotiators when our doctor's in the wrong, but won't back down when he's right."

That approach pays off. "According to the most recent report I've seen from the General Accounting Office," says Larry Rogers, PIE president and CEO, "in 1984, about 57 percent of medical-malpractice claims were closed without payment. Through 1988, we've closed an average of 78 percent of our cases without a dime changing hands. And it's my understanding that, without including defense costs, St. Paul Fire and Marine Insurance Co.'s 1988 average gross payout for cases closed in Ohio with payment was $52,500. Our comparable figure was about $10,000 below theirs. That's partly why we can sell an OBG specialist in Ohio—an industrial state that ranks among the most litigious—$1.2 million in coverage for just $26,400."

The unique marriage of PIE and JMT&K has been so successful that the carrier has expanded into five other states: Indiana, Kentucky, Maryland, Missouri, and West Virginia. Where PIE goes, there goes JMT&K, with nine branch offices to date. The firm has 65 trial attorneys, and may well be the nation's largest devoted well-nigh exclusively to medical-malpractice defense.

Could the insurer-defender symbiosis, if duplicated by other doctor companies, make a significant contribution to reducing malpractice litigation nationwide? An up-close look at how JMT&K operates may help to answer that question.

The winning firm's four founders at Cleveland's 8th District Court of Appeals (from left): Jerome S. Kalur, Aaron Jacobson, James M. Tuschman, and Robert Maynard

Every lawyer develops a medical specialty

"Our firm's lawyers read more medical books than law books," says PIE Vice President Gerard C. Opgenorth, himself a veteran defense attorney. Robert Maynard explains: "New cases are discussed at our weekly staff meeting, so that every lawyer is familiar with every case. But we assign cases to our attorneys according to medical specialty. They're well-versed in their fields, so they don't have to reinvent the wheel with each case."

Last year, the firm's OBG specialist, attorney Jerome S. Kalur, who had won 16 consecutive brain-damaged baby cases, faced one of his toughest challenges when he defended a GP who'd attempted a midforceps delivery that ended in a Caesarean section and a severely brain-injured baby. Recalls Kalur: "I didn't think the doctor had caused the damage, but our position was weakened by the fact that he didn't have midforceps privileges. Based on that departure from the standard of care, our doctor panel voted to settle, and, since the hospital was also involved, a combined sum of $1.5 million was offered. Plaintiffs turned us down flat.

"I wanted to depose the doctors who'd been involved in the mother's care during her hospitalization, but the attorney for the plaintiff baby insisted it would violate the mother's physician-patient confidentiality. That privilege would terminate automatically when her medical records were introduced at the tail end of the plaintiff's case. Meanwhile, I was in the no-win position of having to tell the jury, 'It couldn't have been the midforceps,' without offering them another reasonable brain-damage theory."

Fortunately, the plaintiffs rested their case on a Friday afternoon, giving JMT&K time for a weekend rally. "Twenty minutes later," says Kalur, "I was in the hospital pathologist's office with an order permitting me to view the mother's placental slides." Meconium staining had been charted, and Kalur had a hunch that fetal distress had begun long before the for-

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Constipation, which is easily managed in most patients, is the most commonly reported side effect of Calan SR

BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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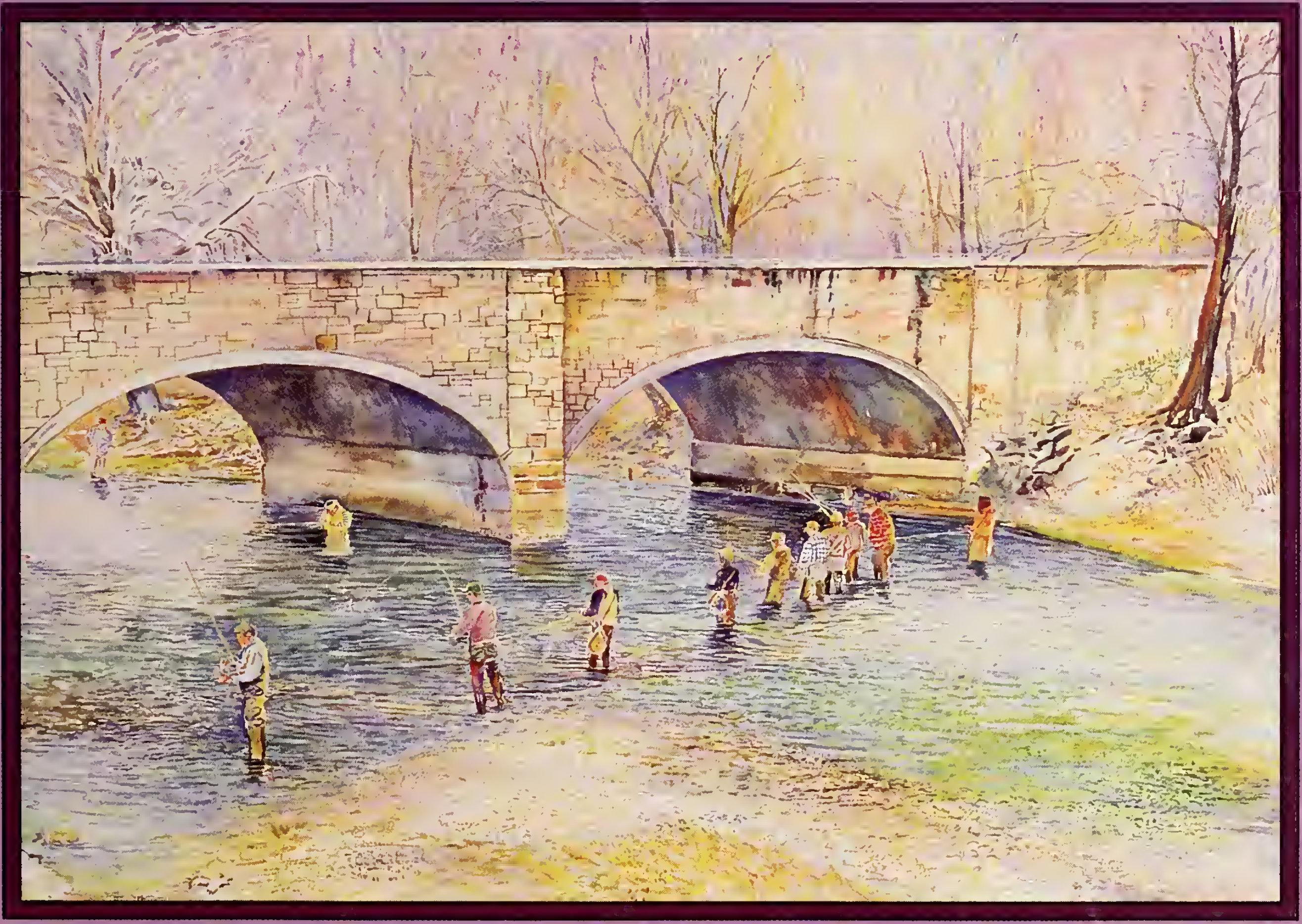

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- ***Breast Conservation Therapy - The St. Luke's Hospital Experience***
- ***The Neurological Aspects of Acquired Immune Deficiency Syndrome (AIDS) - A Clinical Review***

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Owned and edited by the Missouri State Medical Association. *Missouri Medicine, Journal of the Missouri State Medical Association,* is published monthly at 113 Madison, Jefferson City, Missouri 65102. Second-Class postage paid at Jefferson City, Missouri and at additional mailing offices. **Postmaster:** Send address changes to Missouri State Medical Association, c/o Membership Dept. P.O. Box 1028, Jefferson City, MO 65102.

The subscription price per year for non-members is $20.00. The subscription price for members is included in the annual membership dues. The International subscription price is $40.00. Single copies are $4.00 and the cost of the annual August Directory issue is $15.00.

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Printed by the Ovid Bell Press, Inc., Fulton, Missouri 65251.

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MONETAMED PROFILE

THOMAS P. DOYLE, M.D.

HOME: Springfield, Missouri.

AGE: 36.

SPECIALTY: Orthopedic Surgery.

MEDICAL COLLEGE: University of Missouri, 1978

PRACTICE: "I recently purchased an established but declining sole practitioner office from a retiring physician. It's a risk, but they say that great deeds are usually wrought at great risk."

TYPICAL DAY: "Let the horses out of the corral at dawn and maybe even go for a ride. Then, it's surgery if scheduled and a day of torn ligaments and sprained ankles. The evening finds me on the sidelines of one of our high school sports teams."

HOBBIES: Horseback riding and golf. "If I combined activities, would it be polo?"

LAST BOOK READ: Missouri: Images of Nature, photographed by Charles Gurche.

LATEST ACCOMPLISHMENT: "Testing a protective knee pad I'm developing for use by football players."

QUOTE: "Physicians are being deterred from trying new procedures and therapies in patient care. We need to cut the chains and encourage individual innovation."

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Journal of the Missouri State Medical Association

Vol. 89, No. 3 March 1992

This Month's Cover:

March 1st is the opening day of Trout Season in Missouri. Terry Martin, art instructor at William Woods College in Fulton, Missouri, reminds us of the pleasures and challenges associated with fast running water, fishing tackle and hungry, yet wary, game fish.

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NEW OPPORTUNITIES

by J. Regan Thomas, M.D., Editor

Congratulations to Donald Sessions, M.D., and those who worked with him during his tenure as Editor for *Missouri Medicine.* The Journal has continued to grow, improve, and under Dr. Sessions leadership, to innovate. It is that spirit of innovation that now exemplifies *Missouri Medicine.*

New names will be appearing in association with the activities of the Journal. Dennis Weiser, M.S., who recently assumed the responsibilities of Managing Editor, is now settled in and already implementing a number of management goals he set when joining the editorial team. Angela Allen, comes to us with excellent credentials and will be working as Editorial Assistant. Ramona Chapman, M.D., has agreed to continue as Associate Editor and I look forward to working with her.

You will notice changes in the "look" of the Journal. Technical improvements in how our printer processes the Journal will significantly enhance the quality of our printing. We will also be changing some design styles and importantly the type style. This will not only add to the attractiveness of the Journal, but also will improve its readability.

The Letters to the Editor section within the Journal will be expanded. I encourage all of you to utilize this forum. Your views are critical to the direction of the Journal and important to our fellow Missouri physicians.

I frequently will be inviting guest editorials from knowledgeable and perhaps controversial authors. My goal is to use this new opportunity for *Missouri Medicine* to have comment and input from individuals with unique expertise of interest to Missouri physicians. Again, your response and feedback is anticipated and will be welcomed. We hope to not only inform you of timely information but to stimulate your thoughts on current topics in medical practice.

Finally, my opportunity to serve you as Editor provides a natural period of change for the Journal. These changes are actually "new opportunities" for *Missouri Medicine* to achieve a two-fold goal. The first is to provide a publication which may further enhance and strengthen the image of Missouri physicians. The second, is to provide a timely, credible source of useful information for our community of readers. All of us at *Missouri Medicine* look forward to pursuing these "new opportunities" with you.

LETTERS TO THE EDITOR

Dear Dr. Sessions:

It is, I feel, a sad state of affairs when a physician who has devoted fifty years of his life to the field of Medicine is thrust from the roll of medicine because of circumstances beyond his control. I know that I am one of many who are facing the same dilemma, due to the new Missouri Statutes recently enacted. I personally concur that practicing physicians should make every effort to secure continuing medical education in their field, in order to keep their knowledge current in their field. And that local medical organizations should make this material readily available to them, as well as to those outside their own local purview, even to those such as I, *who are unable to travel because of a handicap which limits their mobility.* They could well be supplied by mail or local courier.

I am entering my eleventh year of limited mobility, which prevents me from walking for more than a short distance and precludes climbing or descending stairs. It is a congenital peripheral neuropathy, a condition I had never even heard of during my years of practice. In addition, my spouse of 45 years died a horrible death of cancer during the past year. This necessitated my moving into a home filled with others of such age and limited mobility.

The failure to obtain CME during this period has proved to be my downfall. Like a soldier in the French Foreign Legion, I will be stripped of my brevet. We build miles of ramps, and depressed curbs for the physically handicapped, but those bastions of education, our hospitals, medical schools, and association cannot even provide a few VCR tapes, or brochures to handicapped members who could truly use these services. I would like to continue to learn about medicine, especially in my own field (radiology) if such things were available, even at reasonable cost.

I have no lobby in Jefferson City, or elsewhere as do many others, including the above named, and I cannot join the herd of those who stop their practice for such activity. I may be disabled, but my interest is still alive and well. I would like to retain my license even though I do not, nor will I ever join the active ranks, but the ability to write a prescription for myself, or for my children, as well as the right to talk to others in the domicile which I inhabit as to choice of doctor, whether generalist or specialist (which will be denied me under present laws) would at least let me keep my dignity as a member of a very noble profession.

I have taught for more than twenty-five years at a medical school (no stipend but a title). Innumerable X-ray technologist have been trained by my efforts. As I noted in a previous editorial, my basket has been woven, and my turn to be thrown over the cliff is at hand. I think it unfair.

Clyde M. Witt, M.D.
St. Louis

Dear Dr. Sessions:

In response to Mr. Tromans "justification" for the "Renewal Fee - The cost of Your License As A Professional," *Missouri Medicine*, January 1992, Volume 89, No. 1, page 19. His first statement is correct in quoting what the law does require as far as return of excess moneys from the healing arts fund. My concern is that if the profession, in combining his other two points of logic, were to have to pay for all complaints regardless of merit, then the complaining public could drive medicine out of practice. All one would require is that twenty-five thousand people, which is not an impossible number to assemble in a complaining atmosphere, were to complain about their physicians, then the cost of investigating those twenty-five thousand allegations of individual complaints would have to be borne by the practicing physicians in the state through the licensure fee process. This system, if carried to its illogical conclusion by the Nader mentality, would be totally nonproductive although, under what Mr. Tromans has presented, perfectly legal.

I doubt that there are twice as many problems as reported in his table of statistics but in reality there are twice as many complaining persons. It is my personal judgment that the funding going from a $10.00 fee to a $120.00 annual renewal fee only feeds the process of additional investigation, additional personnel being hired, and additional computer systems being put on line to justify the existence of the high fee.

In Mr. Tromans closing paragraph, "We regard the task of maintaining the public's faith in the quality standards of the profession as being a very important task." What Mr. Tromans is proposing is that we should buy the public's trust by over-funding a state agency for licensure and renewal of licensure to encourage more investigative activity by this group which would mean that we would also encourage more persons to register complaints. I fail to see that it is the obligation of the practicing physicians to encourage this type of activity. If I have misunderstood the point of Mr. Tromans article, I would appreciate his reply in an ensuing issue of *Missouri Medicine.*

Robert N. Miller, M.D. , St. Louis

Dear Dr. Sessions:

I and my partners wish to strongly agree with Dr. Arthur Gale in his article in the January 1992 issue of *Missouri Medicine.* I would like to point out that Mr. Frederick A. Tromans' article, written on the next page, is highly inaccurate. I discussed this with Dr. Joseph Ketcherside, M.D. who informed me that these complaints are not consumer complaints as stated by Mr. Tromans, that most of the complaints, probably at least a third, are simply made by the agents of the BNDD. I believe this information should be verified by yourself and clearly stated in the next issue of *Missouri Medicine.*

David E. Wilson, M.D., Jerry A. Kirkland, M.D.,
Jeffrey A. Luerding, M.D., Paul R. Torres, M.D.,
L. L. Hayes, M.D., Martin A. Kanne, M.D.,
and William M. Gilbirds, M.D., Kansas City

Dear Dr. Sessions,

I am sorry to hear that you will be leaving the job as Editor of *Missouri Medicine*. You have done an outstanding job.

I particularly liked your last editorial, "Will we be all right?" I have given a lot of thought to this recently. There is no doubt that medicine is a noble profession. Unfortunately it is being turned into a business which does not primarily consider the doctor-patient relationship. I hope you are right.

C. Keith Whittaker, M.D., Kansas City

Dear Editor:

I appreciated the summary article on the Utilization and Review changes MSMA pushed firmly for with respect to the "innovative" changes of the Department of Insurance.

As one the individuals who was strongly supportive of this, I know you feel as we do in the Missouri Society of Internal Medicine that this "innovative" activity of the Department of Insurance in further proof that this department needs to have an outside review performed by an objective agency capable of performing this review without regard to political pressures. Their innovative structure changes, or attempts thereof, further documents that this in not a division that is consumer oriented, but rather is self-serving to the industry to protect them from investigative activities that might highlight the consumer abuses which all of us know are rampant in this state.

Thanks again for your assistance and, if we can be of benefit in pursuing this at any time in the future that is necessary, please do not hesitate to contact us.

D. Douglas Westhoff, M.D., Jefferson City MO

Dear Editor:

I read an article recently in the January 1992 St. Louis Metropolitan Medicine Journal by Dr. David L. Wilkinson, President of the Missouri Healing Arts Registration Board. In the article he states, "the proposed budget for 1992 will include approximately $1,150,000 for salaries, $500,000 for non-salary items, and a projected $375,000 for legal support".

Enclosed is a copy of the salaries for Healing Arts Board members for 1991-1992, including that of Dr. Clarence C. Reynolds for $76,421, and Dr. Salvatore N. Riggio for $53,495.

Is it possible to have the 1992 proposed budget breakdown of $2,025,000 published, and is this a usual or a large sum of money for a Registration Board's budget?

John W. McKinney, M.D., St. Louis

Dear Dr. McKinney,

Missouri Medicine does not have access to any individual's proposed salary, but this table was prepared to help you compare the Division of Registration / Board of Healing Arts' personal services expenses (salaries) in terms of actual expenses and appropriated amounts. These expenses, combined with the proposed $500,000 non-salary items, reflect a projected budget total of $1,674,000 for fiscal 1993 that will begin July 1, 1992. Budget figures for legal support are included under Professional Services, which is a part of the Expenses and Equipment section of the Board's annual budget. A review of the budget shows that the amount requested for legal services for 1992, $110,000, does not correspond with Dr. Wilkinson's comment.

This financial information is not complete and interested individuals are encouraged to study the full public financial records rather than accept these excerpts as representative of the Board's financial condition or budgeting procedures.

PERSONAL SERVICES EXPENSES: BUDGET YEARS 1988-92

Budget year Is July to June	1988	1989	1990	1991	1992
Actual Expenses(1)	$465K	$677K	$741K	$762K	
Appropriated Budget Amount(2)	$498K	$715K	$749K	$818K	$1,149K

(1) Partial expenses - all figures rounded to nearest $1,000.
(2) Budget amounts appropriated are subject to revision.
SOURCE: Published Missouri State Budget Information.

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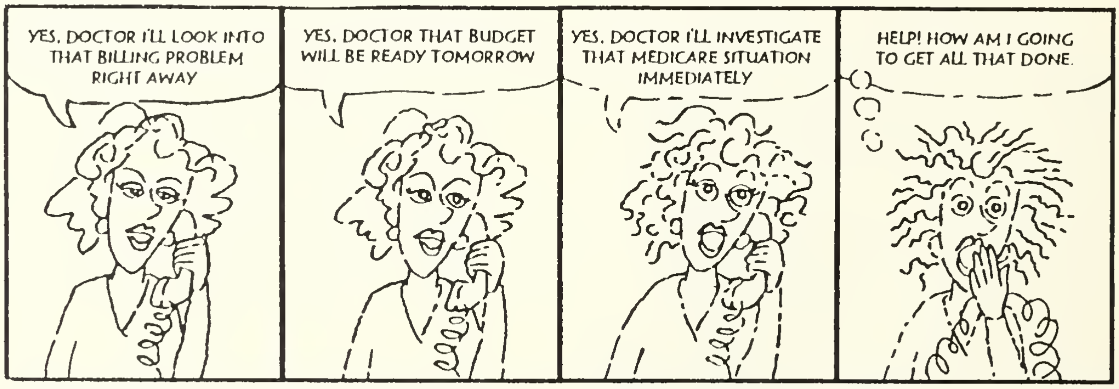

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YES, DOCTOR I'LL INVESTIGATE THAT MEDICARE SITUATION IMMEDIATELY
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WHAT IS A RESPONSIBLE PHYSICIAN?

by
Dorothy D. Reister, M.D.
President, Missouri State Medical Association

The definition of responsibility is the state of being accountable; that for which one is answerable; ability to meet obligations and a duty or trust.

We are accountable to ourselves, our families, our fellow physicians, our churches and the law. These are duties taken voluntarily and they are discharged with pleasure and gratification. There are other duties which are ancillary to these – such as meetings that are required of us in our hospital and clinic affiliations, and, yes, even PTA meetings and school plays, for example.

Yet we as physicians have taken upon ourselves a grave responsibility – that of caring for our patients; we have been entrusted with this obligation. This duty encompasses life to death, well to sick, and it applies in the office, home or hospital, day care nursery or long term care facility. It includes helping patients discuss their medical needs and how they want their health care dollar spent. This charge includes discussing those difficult to discuss end-of-life problems.

Meeting this duty takes time and compassion. Time spent in trying to educate patients and time to communicate. Remember, communication is a two-way street.

Where is responsibility learned? For many of us, it has been learned in the home, but much more family time needs to be spent in activities to encourage such virtue. Fortunately, responsible parents can train their "twigs" to grow into responsible "trees".

Others learn at the fortunate intervention of a relative, friend or teacher who saw that it was lacking in our character. Sometimes it is learned at the hands of the law.

Fortunately, duty and responsibility are vital parts of most physicians' makeup, but some of us shun our duty and leave many jobs to be done by others.

There are forces attempting to divide and conquer the profession. One of the more dangerous of these is the bloc of un-caring, non-contributing physicians who say, "We don't need the MSMA or the AMA," or "What have you done for me?". They also say, "Let George Do it". These people don't read and realize the successes of our negotiations with HICFA and the HHS. The rights of the physicians and their patients have been our primary concern. All of our association's activities require people, however. Every one of us can find some time for volunteer, organizational or political duties.

Another facet of the responsibility issue is a willingness to turn one's face from less than honest practices. We are irate about the Saving & Loan failures, some brokerage houses practices, but has anyone inquired into the insurance companies? It's difficult to explain to patients the news about hospital figures, especially if they have just left a hospital. Seeing hospital comparisons in the newspapers is a similar problem. Perhaps some sense of responsibility can be brought to these industries, too.

In these harsh economic times and in a political arena that is calling for health care cost containment, the American people are being called to make some very tough decisions. We can expect a decline in physician reimbursement and we will need to look at the possibility of some rationing of care in some manner. When elected officials, social engineers, the media, lawyers, representatives of government agencies and others with a stake in health care gather to make those decisions we must make sure that representatives of the medical profession also are at that table.

Our sense of responsibility and our code of ethics does not allow us to turn away from this duty. Let's review quickly what our AMA Principles of Medical Ethics has to say about our duties as physicians.

Continued on page 144

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Continued from page 143

Preamble: The medical profession has long subscribed to a body of ethical statements developed primarily for the benefit of the patient. As a member of this profession, a physician must recognize responsibility not only to patients, but also to society, to other health professionals, and to self. The following Principles adopted by the American Medical Association are not laws, but standards of conduct which define the essentials of honorable behavior for the physician.

I. A physician shall be dedicated to providing competent medical service with compassion and respect for human dignity.

II. A physician shall deal honestly with patients and colleagues, and strive to expose those physicians deficient in character or competence, or who engage in fraud or deception.

III. A physician shall respect the law and also recognize a responsibility to seek changes in those requirements which are contrary to the best interests of the patient.

IV. A physician shall respect the rights of patients, of colleagues, and of other health professionals, and shall safeguard patient confidences within the constraints of the law.

V. A physician shall continue to study, apply and advance scientific knowledge, make relevant information available to patients, colleagues, and the public, obtain consultation, and use the talents of other health professionals when indicated.

VI. A physician shall, in the provision of appropriate patient care, except in emergencies, be free to choose whom to serve, with whom to associate, and the environments in which to provide medical services.

VII. A physician shall recognize a responsibility to participate in activities contributing to an improved community.

I do not apologize for my philosophizing. It worries me to see the decline in taking responsibility for oneself from childhood into adulthood. We are adults and it's time we stood to be counted. Let us stand and be counted as responsible physicians.

◆

Answers to last month's puzzles:

Why is the North Star always in one place?
It isn't. But, the earth's north rotational axis very nearly points to it as we zip together through the universe and it looks stationary.

If the temperature rises from one degree to two degrees, is it twice as warm?
No, unlike weight and height, outdoor temperature does not begin with zero. It has merely risen one degree.

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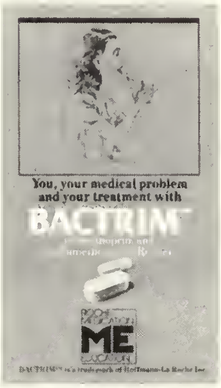
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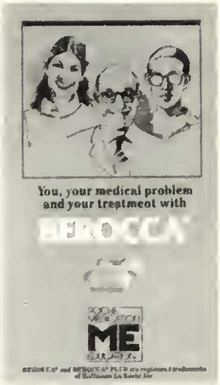
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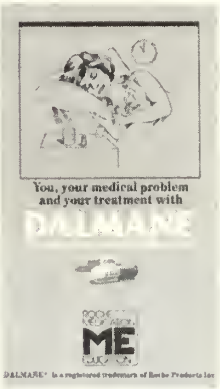
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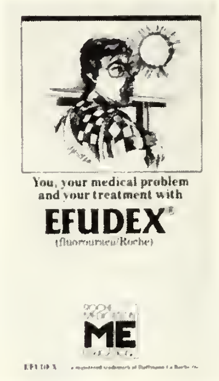
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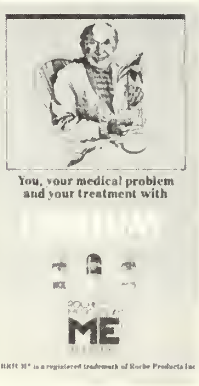
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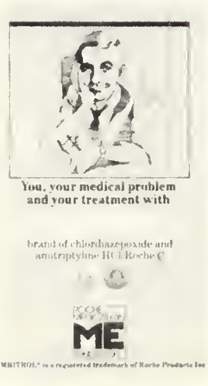
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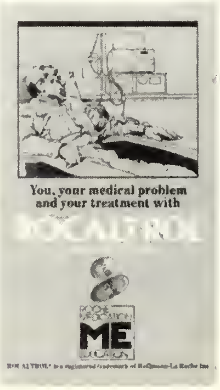
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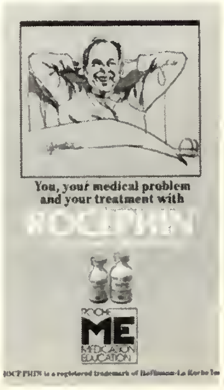
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Dear Janice K. Woerth, Ph.D.

I understand that many CPT codes have been changed in 1992 and that Medicare has also changed its reimbursement methods. I am a non-participating physician with Medicare. How do I know how much to charge for these new CPT codes?

Dr. M.F.

Dear Dr. M.F.,

In December, Medicare sent to all physicians (both participating and non-participating) a Disclosure Report. This Disclosure Report replaces the Medicare Profile that was sent to all physicians in previous years. The Disclosure Report lists the CPT codes, the Par fee schedule, the Non-Par fee schedule, and the Limiting Charges. The amounts listed for each service represent the fee changes brought about by the Resource Based Relative Value Scale (RBRVS).

The list of CPT codes on the Disclosure Report includes the new 1992 evaluation and management codes that replace the 1991 codes for office visits, nursing home visits, hospital visits, consultations, etc. The Par fee schedule is the amount for each service that Medicare allows when the service is rendered by a participating physician. The Non-Par fee schedule is the amount for each service that Medicare allows when the service is being rendered by a non-participating physician. Finally, the Limiting Charge is maximum fee for each service that a non-participating physician can charge a Medicare patient.

As a non-par (non-participating) physician, you are allowed to bill the Limiting Charge listed on the Disclosure Report for the service(s) you render to a Medicare patient. The patient should pay for the entire Limiting Charge (preferably at time of service) and Medicare will reimburse the patient 80% of the Non-Par fee schedule for the service rendered.

Dear Dr. Woerth,

I am confused as to when I should be charging for critical care. Would you please explain how to use the critical care codes?

Dr. R.T.

Dear Dr. R.T.,

According to the 1992 CPT book, critical care is the emergency treatment of patients with acute, life-threatening conditions that require the constant attention of a physician. Acute, life-threatening conditions include cardiac arrest, respiratory failure, bleeding, shock, and postoperative complications. Emergency treatment are services like CPR, electrical conversion of arrhythmia, cardiac output measurement, management of mechanical ventilation, endotracheal intubation, control of internal hemorrhaging, and blood gas monitoring, performed on critically ill patients.

The new critical care codes are found on page 32 of the 1992 CPT book. The code 99291 is for the first hour of critical care given and can only be used once a day. Additional critical care given to a patient during a day is recorded in 30 minute segments using code 99292. Thus, if a patient's condition requires an hour and a half of critical care in the morning and an hour of critical care the same afternoon, the critical care would be reported as follows: 99291, 1st hour of critical care and 3 units of 99292, critical care each additional 30 minutes. Notice that total duration of critical care for the day is added together and reported.

If a physician performs other service or procedures for the patient that do not fall under the definition of critical care management, these services and procedure should be reported separately. For example, a patient who suffers from cardiac arrhythmia is stabilized and then treated by percutaneous transluminal coronary angioplasty, the critical care codes would be used to report the time spent stabilizing the patient and the angioplasty would be reported with a separate code.

Critical care can be performed in a variety of settings, as the intensive care, coronary care, or respiratory care units and the emergency department. The use of critical care codes is not limited to these locations. However, just because the patient is in one of the above mentioned locations, it does not mean the care given qualifies as critical care. Non-critical care given to patients in critical care units is reported using the subsequent hospital care codes (99231-99233).

Continued on Page 148

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Practice Management

Continued from page 148

Do you have a question for Dr. Woerth? Include your questions along with your name, address and phone number and send them to:

Janice K. Woerth, Ph.D.
Practice management
c/o *Missouri Medicine*
P.O. Box 1028
Jefferson City, MO 65102
Or call her direct:
202/363-1780

Although your name will not be used in *Missouri Medicine,* please include your name and phone number so that Dr. Woerth may contact you if she needs additional information to answer your question. ■

MSMA Patient Brochure Information

MSMA has available several patient information brochures. Topics range from breast cancer treatment alternatives to health care cost containment. You may preview sample copies free of charge before ordering. Submit orders and sample requests, by title, on your professional letterhead, to MSMA Brochures, P.O. Box 1028, Jefferson City, MO 65102. Or, you may fax your orders to the MSMA office at 314/636-8552.
Here are just a few of the titles:

A WOMAN'S CHOICE. Presents various breast cancer treatment alternatives in clear layman's language, discussing advantages and disadvantages of each treatment choice. *Cost: 15 cents each.*

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MSMA Preliminary Program

April 9 - 12, 1992
134th Annual Convention
Adams Mark, St. Louis

The Missouri State Medical Association is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

The Missouri State Medical Association designates this continuing medical education activity for Category I credit hours of the Physicians Recognition Award of the American Medical Association.

Although every effort has been made to obtain accurate information, the material in this Preliminary Program is subject to change.

Scientific Program

(listed alphabetically by sponsoring specialty)

ALLERGY

Objectives:
***Morning Session:** To clarify the importance of inhalant allergy in the pathogenesis of asthma, and the importance of treating the underlying causes of asthma. To underscore the emerging awareness that asthma is not often "outgrown."*
***Afternoon Session:** To explore the impact of psychological factors on individuals with asthma. To increase awareness of newer methods for management of asthma.*

Friday, April 10

6:30 p.m. Dinner

Saturday, April 11

7:30 a.m. Continental Breakfast

8:00-9:30 a.m. Scientific Program

Annual Charles A. Brasher, M.D., Memorial Lectureship

"Update on Asthma Therapy"
Speaker: Elliot F. Ellis, M.D., Chief, Division of Allergy and Immunology, Nemours Children's Clinic, Jacksonville, Fla.

Moderator: Dennis Pyszczynski, M.D., Kansas City

co-sponsored by the Missouri Thoracic Society and the American Lung Associations of Eastern and Western Missouri

"Asthma as an Allergic Disease"
Speaker: Benjamin Burrows, M.D., Professor of Medicine, University of Arizona College of Respiratory Sciences Center, Tucson, AZ

"Trends in Asthma Treatment"
Speaker: Paul M. O'Byrne M.D., McMaster University, Hamilton, Ontario, Canada

"What Happens to Asthmatic Children When They Grow Up?"

12:30 p.m. Business Meeting/Luncheon

2:15-3:15 p.m. Scientific Program

"The Role of Psychological Factors in the Outcome of Asthmatics"

"New Directions in the Delivery of Asthma Care: Practical Aspects"
Speaker: Phillip E. Korenblat, M.D., Professor of Clinical Medicine, St. Louis

Moderator: Michele E. Kemp, M.D., Chesterfield

Sponsored by the Missouri State Allergy Association

ANESTHESIOLOGY

Objectives: To provide an understanding of the theory and practice of pain management techniques applicable for the practicing anesthesiologist.

Saturday, April 11

7:00-8:30 a.m. MSA Resident Breakfast

8:30-12:00 noon Scientific Program

"Neuropathic Pain"

"Cancer Pain Management"

"Pharmaco - Therapeutics of Pain Management"

12:00 noon Luncheon

2:00-4:00 p.m. Scientific Program

"Workshop on Anesthetic Techniques of Pain Management"

"Diagnosis and Treatment of Reflex -
Sympathetic Dystrophy"

Moderator: Alice Otto, M.D., St. Louis

Sponsored by the Missouri Society of Anesthesiologists

DERMATOLOGY

Objectives: To teach the current concepts of: the molecular basis of fibrotic skin diseases (morphea, scleroderma, etc.), principles of therapy of fibrotic skin diseases, microscopic and molecular pathology of elastic tissue (aging skin), pemphigus and its subtypes, optimal use of clinical immunology to diagnose bullous disease, the clinical and immunological subsets of lupus erythematosis, interpretation of serologic testing in rheumatic disease, pulse dye lasers and their applications in dermatology, medications and applications of microsurgery in dermatology, clinical management problems in pediatric dermatology, photobiology in dermatologic disease, cutaneous T-cell lymphoma — diagnosis and management.

Friday, April 10

1:00-4:00 p.m. Scientific Program

"Photobiology: Its Role in Dermatologic Disease"

"Cutaneous T-cell Lymphoma: Current Concepts in Diagnosis and Management"

"Dermatologic Surgery I: Microsurgery in the '90s"
Speaker: Lee S. Portnoff, M.D., St. Louis

"Dermatologic Surgery II: Laser Technology and Application"
Speaker: George J. Hruza, M.D., Cutaneous Surgery Center, St. Louis

Moderator: D. Bradley Marrs, M.D., St. Charles

4:00-5:00 p.m. Board of Directors Meeting

Saturday , April 11

8:30-12:00 Scientific Program

"Three Types of Pemphigus and The Optimal Use of the Modern Clinical Immunology Lab"
Speaker: Grant J. Anhalt, M.D., Associate Professor of Dermatology, Johns Hopkins University School of Medicine, Baltimore, Maryland
"Fibrotic Skin Disease/Elastic Fiber Disease and Aging Skin"
Speaker: Jouni J. Uitto, M.D., Ph.D., Chairman, Department of Derma-

tology, Thomas Jefferson Medical College, Philadelphia, Pa.

Moderator: D. Bradley Marrs, M.D.

1:30-5:00 p.m. Scientific Program

"Problems in Pediatric Dermatology Diagnosis and Management"

"Clinical and Immunologic Subsets of Lupus and Interpretation of Serologic Testing in Rheumatic Disease"
Speaker: Richard D. Sontheimer, M.D., Professor of Dermatology Department of Dermatology, University of Texas Southwestern Medical Center, Dallas, Texas

Moderator: D. Bradley Marrs, M.D., St. Charles

7:00 p.m. Cocktails and Dinner

Sponsored by the Missouri Dermatological Society, Inc.

EMERGENCY PHYSICIANS

Saturday, April 11

9:00-10:30 p.m. Scientific Program

"The Role of the Gender Bias in the Evaluation of Chest Pain in the Emergency Department"
Speaker: Larry Lewis, M.D.,Webster Groves

"Soft Tissue of Foreign Bodies"
Speaker: Mark Steele, M.D., Associate Professor of Emergency Medicine, University of Missouri-Kansas City, Resident Director of Emergency Medicine Programs, Truman Medical Center, Kansas City

12:00-2:00 p.m. Board of Directors Meeting and Luncheon

Sponsored by the Missouri Chapter American College of Emergency Physicians

FAMILY PHYSICIANS

Objectives: To help participants recognize health fraud, quackery, and misinformation, and learn to deal with it effectively. To provide participants with an understanding of the common marketing techniques used by the purveyors of quackery. To alert participants to the dangers that health fraud, quackery, and misinformation can cause. To help participants identify and tap into sources of accurate health information. To stimulate participants to ask specific questions about current health quackery.

Saturday, April 11

7:30 a.m. MAFP Directors Meeting

11:00-12:30 p.m. Scientific Program

"Physician Education"
Speaker: Frederick A. Tromans, Executive Director, State Board of Registration for the Healing Arts, Jefferson City

Moderator: Mark Kasten, M.D., Cape Girardeau

1:30-3:00 p.m. Scientific Program

"Quackery & Medical Fraud"
Speaker: John Renner, M.D., President, Consumer Health Information Research Institute, Kansas City

Moderator: Mark Kasten, M.D., Cape Girardeau

Sponsored by the Missouri Academy of Family Physicians

INTERNAL MEDICINE

Saturday, April 11

1:00-3:00 p.m. Scientific Program

"CPT Coding"

Sponsored by the Missouri Society of Internal Medicine

NEUROLOGY

Objectives: To bring the latest technology in neurological diagnos *tic techniques to the group.*

Saturday , April 11

10:00-11:00 a.m.	Scientific Program
11:00-12 noon	MSNA Annual Meeting, Election of Officers
12:00-2:00 p.m.	Reception

Sponsored by the Missouri State Neurological Association

NEUROSURGERY

Objectives: Attendees should be able to understand the nature and diagnosis of cervical spine instability, relevant cervical anatomy, technique for application of Haid plates, pitfalls of selection and application, and patient outcome.

Saturday, April 11

12:00 noon	Luncheon
	"Lateral Mass Haid Plating for Cervical Spine Instability" Speaker: Regis Haid, M.D., Major, USAF, Wilford Hall Medical Center, San Antonio, Texas
	Moderator: John Oro, M.D.

Sponsored by the Missouri State Neurosurgical Society

OPHTHALMOLOGY

Objectives: To provide an update on glaucoma surgical implants and to acquire central information on statewide ophthalmological care network.

Friday, April 10

6:30 p.m.	Board Meeting and Dinner

Saturday, April 11

8:00-12:00 noon	Scientific Program
	"Economics of a Statewide Ophthalmology Health Plan in North Carolina" *Speaker:* Charles Sydnor, M.D., Founder of North Carolina ExPEC Ophthalmology Network, Burlington, NC
	"Baervel DT — Implant for Glaucoma"
12:00 noon	Luncheon
1:00-5:00 p.m.	Scientific Program

Sponsored by the Missouri Ophthalmological Society, Inc.

OTOLARYNGOLOGY

Saturday, April 11

7:00-8:30 a.m.	Breakfast
9:00-12:00	Scientific Program
	"Smoking Cessation"
	"How RBRVS is Going to Impact on Otolaryngology" *Speaker:* David Thompson, C.P.A., Kansas City

Sponsored by the Missouri Society of Otolaryngology-Head and Neck Surgery

PATHOLOGY

Objectives: Practical use of flow cytometry and immunohistochemistry. Also with referrals to formalin fixed and paraffin imbedded tissue.

Saturday, April 11

9:00-10:00 a.m.	Continental Breakfast
10:00-12:00	Scientific Program
	"Practical Use of Newer Techniques in Pathology"
	Moderator: Gordon Johnson, M.D., Crystal City

12:00 noon Luncheon

1:30-4:00 p.m. Board of Directors Meeting

Sponsored by the Missouri Society of Pathologists

PHYSICAL MEDICINE & REHABILITATION

Saturday, April 11

9:00-12:00 noon Scientific Program

"Effect of Spinal Cord Injury Upon G.I. Tract"

12:00 noon Luncheon

Sponsored by the Central Society of Physical Medicine and Rehabilitation

PLASTIC & RECONSTRUCTIVE SURGERY

Objective: To present timely topics of interest to a broad realm of plastic surgeons and also, since this meeting is in conjunction with the Missouri State Medical Association, topics that would also be of advantage to referring physicians or other surgeons that may be involved with the specific program. With this in mind, the papers are reviewed for timeliness, content and interests.

Friday, April 10

2:00-4:00 p.m. Scientific Program

"Trends in Breast and Nipple Reconstruction"
Speaker: John C. McCraw, M.D., Norfolk, Virginia

"The Gastrocnemius Flap in Lower Extremity Reconstruction"

"Reconstruction of the Thumb"
Speaker: Richard Heimburger, M.D., Columbia

"A New Donor Nerve Graft for Median Nerve Grafts"

"Somatotropic Organization of the Facial Nerve"

"Establishment of Clinical Measurement of Fine Sensory Function in the Blind Population"

Moderator: Richard Heimburger, M.D., Columbia

6:30 p.m. Dinner

Saturday, April 11

7:00-8:00 p.m. Breakfast

8:00-10:00 a.m. Scientific Program

"The Relative Radiolucencies of Breast Implant Filler Materials"
Speaker: V. Leroy Young, M.D., Washington University School of Medicine, Division of Plastic Surgery, St. Louis

"Mammographic Changes in Patients with Breast Implants"

Panel Discussion

"Furlow vs. Van Langenbeck Palatoplasty: A Clinical Study"

"Bilateral Malleolar Reconstruction with a Free Fasciocutaneous Flap"

"Expression of Transforming Growth Factor-Beta in Fetal and Adult Wound Healing in Rabbits"

"Effect of Growth Hormone on Chemexfoliate Wounds — Yucatan Mini-pig Model"

"Nerve Repair with Silicone Neurocuff Prosthesis"
Speaker: Bruce Kraemer, M.D., Assistant Professor of Surgery, Washington University School of Medicine, St. Louis

"Lateral Muscle Plantar Flap"
Speaker: Meredith Payne, M.D., Associate Professor of Plastic Surgery, St. Louis University, St. Louis

Moderator: Eric Swanson, M.D., Kansas City

2:30-3:30 p.m. Business Meeting

Sponsored by the Missouri Association of Plastic and Reconstructive Surgeons

PSYCHIATRY

Objectives: Participants will be better informed about general guidelines for safe prescribing (anti-anxiety medications and pain control medications), and the controlled substance monitoring plan in Missouri.

Friday, April 10

2:00-3:30 p.m. Scientific Program

"Prescribing Controlled Substances: The Missouri Board of Healing Arts Perspective"
Speaker: Frederick A. Tromans, Executive Director, The Missouri State Board of Registration for the Healing Arts, Jefferson City

"Prescribing Controlled Substances: The Bureau of Narcotics and Dangerous Drugs Perspective"
Speaker: Ernest Sjoblom, Director, Missouri State Bureau of Narcotics and Dangerous Drugs, Jefferson City

Moderator: Stuart Munro, M.D., Kansas City

3:30-4:30 Business Meeting

5:30-6:30 p.m. Reception

Sponsored by the Missouri Psychiatric Association

RADIOLOGICAL

Objectives: To enhance the membership's awareness and knowledge of current political and economic issues in radiology.

Saturday, April 11

8:30-9:00 a.m. Continental Breakfast

9:00-10:30 a.m. Nominating Committee Meeting,

10:30-11:30 a.m. Board of Directors Meeting

11:30-1:30 p.m. Board Luncheon

1:30-3:30 p.m. Scientific Program

"Political-Economic"
Speakers: Joan Kelly Horn, Second District Congressman;
Thomas Greeson, J.D., Counsel for American College of Radiology;
H. Richard Grisham, President & CEO, St. Anthony's Hospital, St. Louis; Harvey Tettlebaum, J.D., Counsel for the Missouri Radiological Society

3:30-5:00 p.m. Annual Meeting

5:00-6:00 p.m. Reception

Sponsored by the Missouri Radiological Society

SURGERY

Saturday, April 11

12:45 p.m. Luncheon

9:00-11:00 a.m. Scientific Program

Co-sponsored by the American Cancer Society
Sponsored by the Missouri Chapter, American College of Surgeons

Thursday, April 9

6:00-8:00 p.m. Board of Directors Dinner

Friday, April 8

10:45-12 noon Scientific Program

"Laparoscopic Hernia Repair: Is it a Reality?"
Speakers: Charles Andrus, M.D., St. Louis University, St. Louis *and* Raymond Keltner, Jr., M.D., St. Louis University, St. Louis

1:30-4:00 p.m. Scientific Program

"Trauma and Auto Insurance Reform"
Speaker: Tom Helling, M.D., Kansas City

Moderator: Bryan R. Troop, M.D., St. Louis

Sponsored by the Missouri State Surgical Society

THORACIC

Objectives: 1) Review causes for increases in asthma morbidity and mortality despite therapeutic advances, 2) Summarize NHLBI diagnostic and treatment guidelines for asthma, and 3) Emphasize the importance of a follow-up care plan for the outpatient asthmatic.

Saturday, April 11

11:00-12:30 p.m. Scientific Program

Annual Charles A. Brasher, M.D., Memorial Lectureship

"Update on Asthma Therapy"
Speaker: Elliot F. Ellis, M.D., Chief, Division of Allergy and Immunology, Nemours Children's Clinic, Jacksonville, Fla.

Moderator: Dennis Pyszczynski, M.D.

Co-sponsored by the Missouri State Allergy Association
Sponsored by the Missouri Thoracic Society and the American Lung Associations of Eastern and Western Missouri

Affiliated Annual Program Events

CANCER

Saturday, April 11

9:00-11:00 a.m. Scientific Program

"Tobacco Cessation Training for Health Professionals"

co-sponsored by the Missouri Chapter, American College of Surgeons

12:45-1:45 p.m. American Cancer Society, Missouri Chapter, American Cancer Society Liaison Luncheon

Sponsored by the American Cancer Society

HEART ASSOCIATION

Friday, April 10

2:00-3:30 p.m. Scientific Program

Sponsored by the Missouri Heart Association

WOMEN PHYSICIANS

Objectives: The physician who attends this session will learn about the definitions of sexual harassment, the medical and legal implications of sexual harassment and any medical or legal recourses that the victim may have.

Saturday, April 11

10:30-12 noon Scientific Program

"Medical Aspects of Sexual Harassment"
Speaker: Lillian Gonzalez-Pardo, M.D., President, American Medical Women's Association, Department of Pediatric Neurology, Kansas University Medical School, Kansas City, KS

"Legal Aspects of Sexual Harassment"
Speaker: Mr. Louis Gilden, Attorney at Law, St. Louis

Moderator: Jo-Ellyn M. Ryall, M.D., St. Louis

12:00-2:00 p.m. Luncheon

Sponsored by the American Medical Women's Association

Annual Meetings and Events

Thursday, April 9

10:00-12 noon	**Opening Session, House of Delegates**
12:00 noon	St. Louis Metropolitan Medical Society Luncheon
12:00 noon	Metropolitan Medical Society of Greater Kansas City Luncheon
6:00-7:00 p.m.	**Reception** Honoring MSMA President and Auxiliary President

Friday, April 10

7:00 a.m.	District #1 Breakfast District #2 Breakfast District #5 Breakfast District #6 Breakfast District #8 Breakfast District #10 Breakfast
8:30-11:30 a.m.	MU Alumni Organization Board of Governors Breakfast Meeting
9:00-11:00 a.m.	General Session **Featured speaker:** Peter Huber "Junk Science and Quack Medicine in the Courtroom"
10:30-12:30 p.m.	Resident Physician Section Meeting,
10:30-12:30 p.m.	Young Physician Section Meeting
11:00-12:30 p.m.	Discuss the Issues with your AMA Delegates
11:30-12:30 p.m.	Physicians Health Committee Scientific Program
12:00 noon	District #7 Luncheon District #9 Luncheon Student Delegates Luncheon
12:30-2:30 p.m.	MU Alumni Awards Luncheon
1:30-4:00 p.m.	Young Physicians / MOMEDICO Mock Trial
2:00-4:00 p.m.	Hospital Medical Staff Section Meeting
4:00-6:00 p.m.	**House of Delegates**
6:00-7:30 p.m.	UMKC Reception
6:00-7:30 p.m.	St. Louis University Reception
6:00-7:30 p.m.	Washington University Reception
6:30-7:30 p.m.	MU Alumni Reception

Saturday, April 11

7:15 a.m.	MSMA Auxiliary Public Affairs Breakfast
1:00-4:00 p.m.	Resident Physician Section Workshop
1:30 p.m.	MMPAC Meeting
6:00 p.m.	**Presidential Inauguration** *Carol F. Williams, M.D., St. Louis*
7:00 p.m.	**MSMA Reception**

Sunday, April 12

7:30-10:00 a.m.	**Final Session, House of Delegates**

The MSMA Auxiliary presents

The Non-Event Fundraiser
Thursday, April 9, 1992

– No Sit down dinner
– No bidding auction
– No live entertainment

Your tax-deductible contribution will just give you the satisfaction of knowing you're helping the Missouri State Medical Foundation and the AMA Education and Research Foundation.

Yes! I want to contribute to the AMA-ERF or the Missouri State Medical Foundation. My check is payable to AMA-ERF or the MSMA.

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Missouri State Medical Association Financial Statement - 1991

MISSOURI STATE MEDICAL ASSOCIATION

BALANCE SHEETS
December 31, 1991 and 1990

ASSETS	1991	1990
CURRENT ASSETS		
Cash and Cash Investments (Note 2)	$1,329,879	$1,430,066
Marketable Securities (Note 3)	997,166	566,077
Accounts Receivable	11,329	9,416
Other Current Assets	231	231
Total Current Assets	2,338,605	2,005,790
INVESTMENTS		
Common Stock of MSMI, Inc., a 100% owned unconsolidated Subsidiary, at cost plus equity in undistributed net income (Note 4)	35,403	21,760
Common Stock of MOMED Holding Company, a 10.8% owned Affiliate, lower of cost or market (Note 5)	169,295	120,925
Total Investments	204,698	142,685
LAND, BUILDING AND EQUIPMENT (Note 6)	605,555	653,861
OTHER ASSETS, Cash Surrender Value	61,071	47,057
	$3,209,929	$2,849,393

LIABILITIES AND MEMBERS' EQUITY	1991	1990
CURRENT LIABILITIES		
Accounts Payable and Accrued Expenses	$ 23,626	$ 22,297
Due to the American Medical Association	169,845	131,137
Due to County Medical Societies	-0-	776
Deferred Revenue	199,689	163,506
Total Current Liabilities	393,160	317,716
SUBORDINATED NOTES PAYABLE (Note 7)	590,781	590,781
DEFERRED COMPENSATION (Notes 8)	53,841	47,693
Total Liabilities	1,037,783	956,190
MEMBERS' EQUITY	2,172,147	1,893,203
	$3,209,929	$2,849,393

STATEMENTS OF REVENUES, EXPENSES AND CHANGES IN MEMBERS' EQUITY
Years Ended December 31, 1991 and 1990

	1991	1990
REVENUES		
Dues	$1,260,494	$1,294,998
Subscriptions	17,431	19,541
Exhibit Rentals	15,225	24,250
Advertising	59,929	60,038
Commissions on Collections	22,100	23,955
Investment Income (Note 9)	109,131	142,173
Administrative Fees and Office Rent (Note 10)	48,233	45,308
Seminars and Workshops	59,635	25,635
Physicians Rehab Program (Note 11)	83,235	94,771
Other Revenues	29,450	22,945
Total Revenues	$1,704,863	$1,753,614
EXPENSES		
Salaries & Fringe Benefits	630,525	585,258
Office Expenses	108,626	102,879
Travel Expense	10,970	4,353
Commissions & Committee Expense	5,139	722
Depreciation	48,306	35,812
MSMA Auxiliary	6,767	10,766
Journal Expense	120,783	139,512
Meeting Expense	217,597	193,859
Public Affairs	36,453	30,865
Telenet System	-0-	320
Consultant & Professional Fees	57,585	52,527
Public Relations Program	32,710	33,457
Student Awards	1,200	1,200
Tort Reform	10,817	28,504
Miscellaneous	9,982	16,610
Seminars and Workshops	64,981	16,765
Physicians Rehab Program (Note 11)	131,834	110,561
Charity Care Program	-0-	976
Total Expenses	$1,494,275	$1,364,946

The Notes to Financial Statements are an integral part of these statements

MISSOURI STATE MEDICAL ASSOCIATION

STATEMENTS OF REVENUES, EXPENSES AND CHANGES IN MEMBERS' EQUITY - (Continued)
Years Ended December 31, 1991 and 1990

	1991	1990
Excess (deficiency) of revenues over expenses before equity in net income of investments	$ 210,588	$ 388,668
Equity in Net Income (loss) of MSMI, Inc. (Note 4)	13,643	276
Equity in Net Income (loss) of Missouri Medical Insurance Company (Note 5)	48,370	120,925
Excess (deficiency) of revenues over expenses	272,601	509,869
Members' Equity, beginning	1,893,203	1,383,334
Prior Period Adjustment (Note 3)	6,343	-0-
Members' Equity, ending	$2,172,147	$1,893,203

The Notes to the Financial Statements are an integral part of these statements.

Missouri State Medical Association Financial Statement - 1991

STATEMENTS OF CASH FLOW
Years Ended December 31, 1991 and 1990

Cash Flows from Operating Activities:		
Net Income	$ 272,601	$ 509,869
Prior Period Adjustment	$ 6,343	-0-
Adjustments to reconcile Net Income to Net Cash provided by Operating Activities:		
Depreciation	$ 48,306	$ 35,812
(Increase) or Decrease to Accounts Receivables	(1,913)	6,255
(Increase) or Decrease in other Current Assets	-0-	944
(Increase) or Decrease in Cash Surrender Value	(14,014)	(7,980)
(Increase) or Decrease in Accounts Payable	1,329	14,388
Increase or (Decrease) in Due to the AMA	38,708	42,675
Increase or (Decrease) in Due to the County Societies	(776)	(8,617)
Increase or (Decrease) in Deferred Revenue	36,183	35,236
Increase or (Decrease) in Deferred Compensation	6,148	5,942
Total Adjustments	113,971	124,655
Net Cash provided by Operations	$ 392,915	$ 634,524
Cash Flows from Investing Activities:		
(Increase) or Decrease in Marketable Securities	$ (431,089)	$ 14,439
(Increase) or Decrease in MSMI, Inc. Investment	(13,643)	(276)
(Increase) or Decrease in Affiliate	(48,370)	(120,925)
Purchase of Equipment	-0-	(12,530)
Net Cash used Investing	(493,102)	119,292)
Net Increase or (Decrease) in Cash	(100,187)	515,232
Cash at beginning of Year	1,430,066	914,834
Cash at end of Year	$1,329,879	$1,430,066

NOTES TO FINANCIAL STATEMENTS

Note 1. Summary of Significant Accounting Policies

Financial Statements:

The financial statements of the Missouri State Medical Association are prepared on the accrual basis of accounting.

Dues:

Due are assessed to members on a calendar year basis. On December 31st of each year, members delinquent in payment of dues forfeit their memberships. The Association may also collected from members, dues and assessments of the American Medical Association and County Medical Societies, which are then forwarded to the respective organizations by the Association. The Association receives a fee for providing this service. Amounts received from members prior to January 1st of each year, which represents the subsequent year's dues, are recorded as deferred revenue.

Valuation of Accounts Receivable:

Accounts receivable are stated at face amount with no allowance for doubtful accounts. An allowance for doubtful accounts is not considered necessary because probable uncollectible accounts are immaterial.

Marketable Securities:

Investments in marketable equity securities are carried at the lower of aggregate cost or market at the balance sheet date.

Property and Equipment:

Property and equipment are carried at cost. Depreciation is computed by the straight-line method based on estimated useful lives of the related assets.

Income Taxes:

The Association is exempt from Federal Income Taxes under the Internal Revenue Code Section 501(c)(6). Any unrelated business income is subject to income tax.

NOTES TO FINANCIAL STATEMENTS (Continued)

Note 2. Cash and Cash Investments

The balance of cash and cash investments consisted of the following at December 31:

	1991	1990
Petty Cash	50	50
Cash in bank, Executive Secretary Account	9,426	4,308
Cash in bank, Treasury Account	(15,821)	68,320
Cash in bank, Physicians Rehab Program	2,125	1,714
Money Market Fund	149,099	164,674
Certificates of Deposit	1,185,000	1,191,000
	$1,329,879	$1,430,066

Note 3. Marketable Securities

Investments in marketable equity securities are carried at the lower of aggregate cost or market at the balance sheet date.

The following is a summary of marketable equity securities at December 31:

	1991	1990
Aggregate Cost	$ 997,166	$ 566,077
Aggregate Market	1,218,856	594,506
Unrealized gain	$ 221,690	$ 28,429

Net realized gain of $15,514 and $8,969 on the sale of marketable equity securities were included in revenue for the years ended December 31, 1991 and 1990 respectively.

Note 4. Investments in Unconsolidated Subsidiary

The Association's investment in MSMI, Inc., a 100% owned unconsolidated subsidiary, is carried at cost ($2,000) plus equity in the undistributed net earnings since the date of acquisition. At December 31, 1991 and 1990, the investment in the subsidiary is carried at $35,403 and $21,760 respectively.

Condensed financial data relating to the subsidiary follows:

NOTES TO FINANCIAL STATEMENTS

Note 4. Investment in Unconsolidated Subsidiary (Continued)

MSMI, INCORPORATED
BALANCE SHEETS

ASSETS	---December 31--- 1991	1990
CURRENT ASSETS		
Cash, including Savings Account	23,808	8,360
Receivables	1,195	-0-
Total Current Assets	$25,003	$ 8,360
INVESTMENTS - COMMON STOCK	15,000	15,000
	$40,003	$23,360
LIABILITIES AND STOCKHOLDERS' EQUITY		
CURRENT LIABILITIES		
Accounts Payable	$ 4,600	$ 1,600
STOCKHOLDERS' EQUITY		
Common Stock, $10 per value: 3000 shares authorized; 200 shares issued and outstanding	2,000	2,000
Retained Earnings	33,403	19,760
Total Stockholders' Equity	35,403	21,760
	$40,003	$23,360

STATEMENT OF INCOME AND EXPENSES	For the Year Ended ---December 31--- 1991	1990
Income	23,095	6,008
Expenses	9,452	5,732
Net Income	$13,643	$ 276

Missouri State Medical Association Financial Statement - 1991

Note 5. Investment of Affiliate

The Association exercises significant management control over its affiliate, MOMED Holding Company, owner of Missouri Medical Insurance Company (MOMEDICO) a mono-line Medical Professional Liability Insurance Company founded and sponsored by the Association.

At December 31, 1991 and 1990 ownership was:

Total Ownership	Shares Outstanding		Votes Per Share	Votes
Class A C/S - $1 PAR	222,343	x	1	222,343
Class B C/S - $25 PAR	24,185	x	15	362,775
				585,118

MSMI, Inc. (wholly owned subsidiary of Missouri State State Medical Association) ownership

Class A C/A - $ PAR	4,000	x	1	4,000

Missouri State Medical Association ownership

Class B C/A - $25 PAR	24,185	x	15	362,775
				366,775

Combined ownership = 11.43%
Combined voting rights = 62.68%

This investment is carried at the lower of cost or market. Original cost is $322,775 with market value at December 31, 1991 and 1990 of $169,295 and $120,925 respectively.

Most recently available condensed financial data relating to the subsidiary follows:

	---December 31--- 1990	1989
Consolidated Balance Sheets		
Investments	$46,807,860	$41,317,027
Other Assets	3,068,106	2,239,538
Total Assets	$49,875,966	$43,556,565
Reserve for Losses & Loss Adjustment Expense	$32,368,926	$32,217,508
Other Liabilities	11,587,589	8,864,661
Total Liabilities	43,956,515	41,082,169
Stockholders' Equity	5,919,451	2,474,396
Total Liabilities and Stockholders' Equity	$49,875,966	$43,556,565

	---December 31--- 1990	1989
Consolidated Statement of Operations		
Revenues	$15,088,607	$17,079,328
Expenses	10,013,789	12,722,213
	5,074,818	4,357,115
Provision for Federal Income Tax	1,631,991	1,481,843
Earnings (loss) before extra ordinary item	3,442,827	2,875,272
Net Earnings	$ 3,442,827	$ 2,875,272

Note 6. Land, Buildings and Equipment

Consists of the following December 31 (at Cost):

	1991	1990
Land	83,000	83,000
Buildings	581,704	581,704
Furniture and Equipment	293,432	293,432
	958,136	958,136
Less Accumulated Depreciation	352,581	304,275
	$ 605,555	$ 653,861

Note 7. Subordinated Notes Payable

In 1978, the Association formed a wholly owned subsidiary, MOMEDICO, to provide professional liability and premises liability insurance for physicians and support technicians practicing in the State of Missouri. Before obtaining professional liability insurance through MOMEDICO, physicians residing in the State of Missouri were required to subscribe to non-interest bearing subordinated notes based upon the physician's underwriting classification. These notes were issued by the Association and the subscriptions were payable over a three-year period.

The Association was the sole stockholder of MOMEDICO prior to public stock offering of MOMEDICO in 1980. At that time, the requirement to purchase subordinated notes was eliminated with new policies effective after January 31, 1980.

In connection with the public stock offering in 1980, MOMEDICO assumed, in exchange for 100,815 of the 125,000 shares of the Class B common stock the Association held in MOMEDICO, the face value of the subordinated notes issued and outstanding. MOMEDICO also assumed the balance of the installments receivable on the subordinated notes and a bank note payable from the Association. Under an exchange offer made by MOMEDICO, holders of the Association's subordinated note could exchange fully paid notes for Class A common stock at the rate of $20 face value per share.

On October 15, 1980, the Association reassumed all notes not exchanged and also the installments receivable on the unexchanged notes as well as an amount equal to the installments receivable of the bank note payable.

The notes require repayment, only upon the Association's accumulation of in excess of $3,000,000 of members' equity at the end of any calendar year, that the Association has sufficient funds and assets, exclusive of the amount subject to repayment, to permit it to carry on business, and that the repayment be approved by the Council of the Association. Interest may be paid on the notes, but it is at the discretion of the Council of the Administration.

Note 7. Subordinated Notes Payable (Continued)

Redemption of the notes are provided for in the event of death, disability, retirement of the holder, or if the holder has moved both his person and his practice out of the State of Missouri. However, redemption for such causes may be delayed or refused if the Council determines the Association is not on a financially reasonable basis to make such redemptions.

At both December 31, 1991 and 1990, the balance of the subordinated notes payable was $590,781. This amount reflects the face value of the notes reassumed by the Association on October 15, 1980, less the installments receivable on the outstanding notes, and less all redemptions made from 1981 through 1985. In 1985, the Council of the Administration placed a moratorium on the redemption of the subordinated notes.

Note 8. Deferred Compensation Plan

The Association entered into a deferred compensation agreement with its Executive Vice-President on December 31, 1980 which provides for payments upon retirement, death or disability. The agreement also stipulates that a pro-rata settlement of benefits will be made in the event of voluntary termination of employment with the Association, once five years of services is completed after the agreement becomes effective. A similar agreement was enacted May 1, 1990 between the Executive Director and the Association. Both agreements are funded through insurance policies.

At December 31, 1991 and 1990, the Association's liability under the terms of these agreements were $53,841 and $47,693 respectively with the underlying cash surrender value of life insurance being $61,071 and $47,057 respectively.

On December 17, 1989, the Association adopted a Deferred Compensation Plan for Employees pursuant to Internal Revenue Service Code Section 457. Virtually all employees are covered by the Plan. Within Internal Revenue Service guidelines, participants may elect to defer a portion of their income until retirement. Funds invested for these purposes amounted to $34,521 and $11,855 as of December 31, 1991 and 1990 respectively.

Missouri State Medical Association Financial Statement - 1991

Note 9. Investment Income

The following is a summary of investment income for the years ended December 31:

	1991	1990
Interest on Cash Investments	83,470	99,394
Interest on Notes Receivable	-0-	-0-
Dividends	10,146	33,810
Realized Gain or (Loss) on the Sale of Marketable Equity Securities	15,514	8,969
Total	$109,130	$142,173

Note 10. Related Party Transactions

The Association provides administrative services to MSMI, Inc. (a wholly owned subsidiary) and Missouri State Medical Foundation (an affiliate). For providing these services, the Association receives an annual fee from each of the two organizations. For the years ended December 31, 1991 and 1990, the administrative fee assessed Missouri State Medical Foundation was $10,000 and $12,000 and the administrative fee assessed MSMI, Inc. was $4,600 and $4,100 respectively.

The Association, during 1990 also provided office space, office furniture and office services for its affiliate, MOMEDICO. In exchange for providing these services, the Association assessed a monthly fee. For the year ended December 31, 1990 fees were $15,900.

Note 11. Physicians Rehabilitation Program

During 1986, the Association established a Physicians Rehabilitation Program (PRP), the purpose of which is to motivate impaired physicians to voluntarily enter treatment and to provide such a treatment program.

Funding for the program is provided by fees assessed to participants as well as from donations received from supportive individuals and organizations.

Note 11. Physicians Rehabilitation Program (Continued)

The following is a summary of the program's revenue and expenses for the year ended December 31, 1991 and 1990.

	1991	1990
Revenue:		
Hospital Staff	38,195	48,180
Insurance Company	25,760	30,000
Participant Fees	18,255	15,171
Donations	1,025	1,420
Total Revenues	$83,235	$94,771
Expenses:		
Medical Director-Contracted Salary	$54,167	$50,000
Salary - Coordinator and Administrative Assistant	41,208	34,194
Office Expense	13,272	3,471
Secretarial Services	1,121	2,786
Publications	1,531	769
Travel	10,651	9,411
Legal and Consulting Fees	1,297	597
PHC Committee	827	-0-
Bad Debts	-0-	-0-
Facility Expense	6,665	5,174
Lab Fees	153	105
Counselor Services	942	4,054
Other Expenses	-0-	-0-
Total Expenses	$131,834	$110,561

Note 12. Pension Plan

The Association maintains a non-contributory defined benefit pension plan covering substantially all of its employees. Established April 1, 1959, the Plan has a March 31st year end. Eligibility requirements include a minimum of 12 service months and a minimum age of 21. Normal retirement age is 65 with provision for pro-rata benefits prior to 65. Vesting is scheduled over six years of covered service. The Individual Aggregate Actuarial Cost Method is used as the basis for the funding standard account computation. At March 31, 1991, the most recently available actuarial information, fair value of plan assets was $701,429 ($612,676 in 1990). Total benefit obligation was $485,510 ($443,568 in 1990) with a vested benefit obligation of $484,214 (442,355 in 1990).

Note 12. Pension Plan

Employer contributions for FYE March 31, 1991 was $41,189 ($32,561 in 1990). Additional Plan income was $99,110 ($102,585 in 1990) with expenses of $10,417 ($2,182 in 1990) and benefits paid in the amount of $10,281 ($63,565 in 1990), net income for the plan year was $88,693 ($36,837 in 1990). There was no unconditional liabiling for any unfunded accumulated benefit obligation plus any prepaid pension cost, nor unfunded accumulated benefit obligation reduced by any unfunded accrued pension cost. Assets are invested primarily in government securities and corporate stocks and bonds.

Not periodic pension cost of $41,189 ($32,561 in 1990) was composed of employer's normal cost of $37,655 ($30,161 in 1990), amortization charge of $233 ($233 in 1990) and interest of $3,310 ($2,167 in 1990).

Note 13. Prior Year Adjustment

An adjustment of $6,343 increasing dues income for 1990 arose during the course of the current year's audit.

SCHEDULE OF SELECTED EXPENSES
December 31, 1991 and 1990

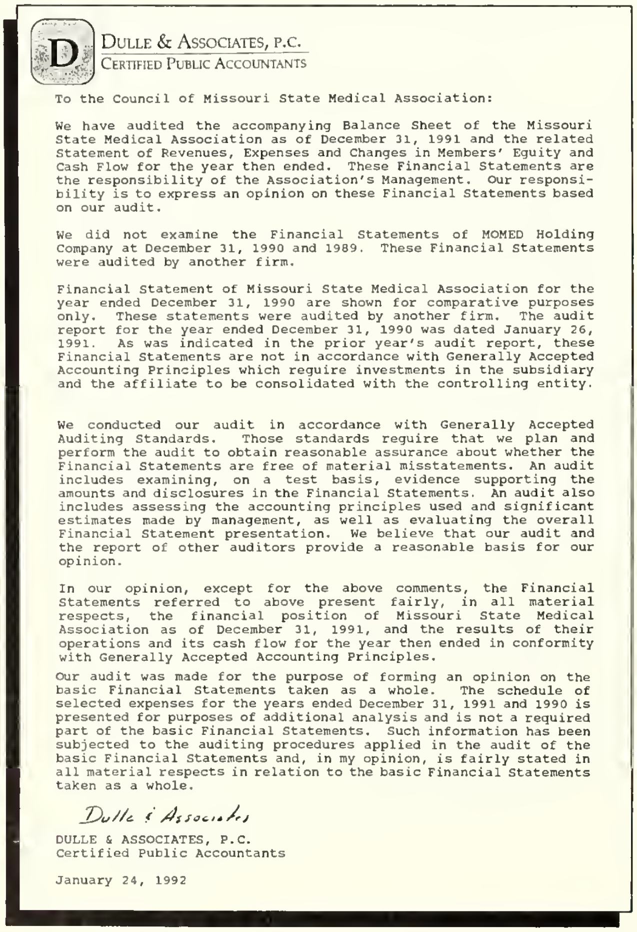
SALARIES AND FRINGE BENEFITS	1991	1990
Salaries	$477,024	$457,614
Payroll Taxes	37,494	37,860
Employees Life & Disability Insurance	3,686	12,483
Employees Hospitalization Insurance	48,014	40,891
Employees Pension Plan Contribution	41,507	30,447
Deferred Compensation Plan Expense	22,800	5,963
Total Salaries & Fringe Benefits	$630,525	$585,258
OFFICE EXPENSES		
Occupancy Expense	18,725	16,009
Office Supplies	22,611	14,133
Computer Expense	11,556	13,371
Postage	17,182	17,564
Telephone	14,101	12,608
Insurance	4,975	5,982
General Expense	(12,590)	-0-
Taxes, Real Estate	4,893	3,572
Custodian Fees	10,017	3,777
Equipment Rental and Repair	7,531	8,466
Dues and Directories	9,625	7,397
Total Office Expenses	$108,626	$102,879
COMMISSIONS AND COMMITTEES EXPENSES		
Commissions:		
Commission on Medical Economics, Third Party Medicine and Governmental Relations	$ 163	$ 73
Commission on Continuing Medical Education	3,819	-0-
Commission on Interspeciality Medicine	253	250
Commission on Medical - Legal Affairs	98	-0-
Total Commissions Expenses	$ 4,333	$ 323

Missouri State Medical Association Financial Statement – 1991

SCHEDULE OF SELECTED EXPENSES (Continued)
December 31, 1991 and 1990

	1991	1990
COMMISSIONS AND COMMITTEES EXPENSES (Continued)		
Committees:		
Committee on Planning	-0-	55
Committee on Legislative Affairs	806	344
Total Committee Expenses	806	399
Total Commissions & Committee Expenses	$5,139	$ 722
MEETING EXPENSES		
Meetings:		
Annual Session	$ 78,708	$ 61,457
Council	46,861	49,780
AMA Delegates' Expenses	82,926	68,859
Resident Physicians Section	1,135	2,528
Young Physicians Section	-0-	2,482
Student Section	2,413	4,600
Discretionary Council Fund	1,116	1,484
Hospital Medical Staff	4,438	2,669
Total Meeting Expenses	$217,597	$193,859

Missouri State Medical Association Financial Statement - 1991



DULLE & ASSOCIATES, P.C.
CERTIFIED PUBLIC ACCOUNTANTS

To the Council of Missouri State Medical Association:

We have audited the accompanying Balance Sheet of the Missouri State Medical Association as of December 31, 1991 and the related Statement of Revenues, Expenses and Changes in Members' Equity and Cash Flow for the year then ended. These Financial Statements are the responsibility of the Association's Management. Our responsibility is to express an opinion on these Financial Statements based on our audit.

We did not examine the Financial Statements of MOMED Holding Company at December 31, 1990 and 1989. These Financial Statements were audited by another firm.

Financial Statement of Missouri State Medical Association for the year ended December 31, 1990 are shown for comparative purposes only. These statements were audited by another firm. The audit report for the year ended December 31, 1990 was dated January 26, 1991. As was indicated in the prior year's audit report, these Financial Statements are not in accordance with Generally Accepted Accounting Principles which require investments in the subsidiary and the affiliate to be consolidated with the controlling entity.

We conducted our audit in accordance with Generally Accepted Auditing Standards. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the Financial Statements are free of material misstatements. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the Financial Statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall Financial Statement presentation. We believe that our audit and the report of other auditors provide a reasonable basis for our opinion.

In our opinion, except for the above comments, the Financial Statements referred to above present fairly, in all material respects, the financial position of Missouri State Medical Association as of December 31, 1991, and the results of their operations and its cash flow for the year then ended in conformity with Generally Accepted Accounting Principles.

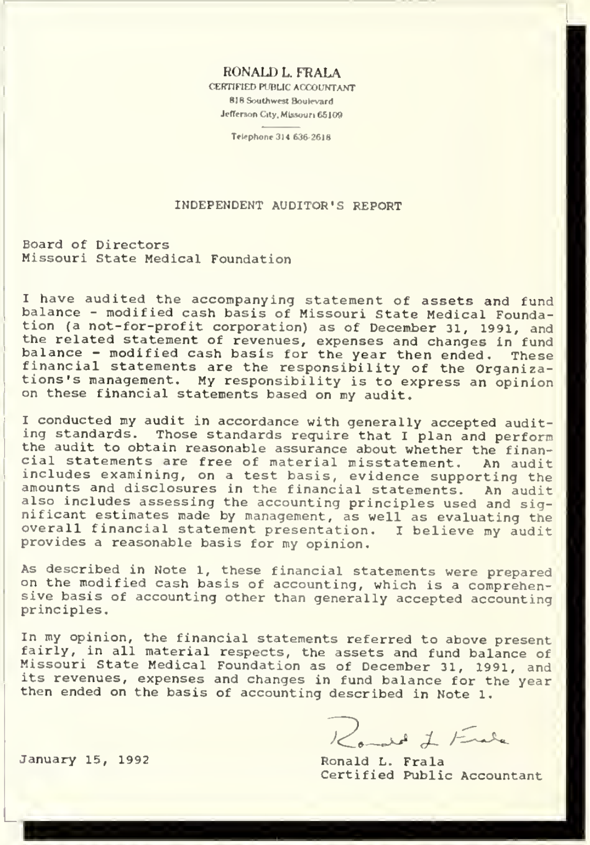
Our audit was made for the purpose of forming an opinion on the basic Financial Statements taken as a whole. The schedule of selected expenses for the years ended December 31, 1991 and 1990 is presented for purposes of additional analysis and is not a required part of the basic Financial Statements. Such information has been subjected to the auditing procedures applied in the audit of the basic Financial Statements and, in my opinion, is fairly stated in all material respects in relation to the basic Financial Statements taken as a whole.

Dulle & Associates

DULLE & ASSOCIATES, P.C.
Certified Public Accountants

January 24, 1992

Missouri State Medical Foundation Financial Statement – 1991



RONALD L. FRALA
CERTIFIED PUBLIC ACCOUNTANT
818 Southwest Boulevard
Jefferson City, Missouri 65109

Telephone 314 636-2618

INDEPENDENT AUDITOR'S REPORT

Board of Directors
Missouri State Medical Foundation

I have audited the accompanying statement of assets and fund balance - modified cash basis of Missouri State Medical Foundation (a not-for-profit corporation) as of December 31, 1991, and the related statement of revenues, expenses and changes in fund balance - modified cash basis for the year then ended. These financial statements are the responsibility of the Organizations's management. My responsibility is to express an opinion on these financial statements based on my audit.

I conducted my audit in accordance with generally accepted auditing standards. Those standards require that I plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. I believe my audit provides a reasonable basis for my opinion.

As described in Note 1, these financial statements were prepared on the modified cash basis of accounting, which is a comprehensive basis of accounting other than generally accepted accounting principles.

In my opinion, the financial statements referred to above present fairly, in all material respects, the assets and fund balance of Missouri State Medical Foundation as of December 31, 1991, and its revenues, expenses and changes in fund balance for the year then ended on the basis of accounting described in Note 1.

Ronald L Frala

January 15, 1992

Ronald L. Frala
Certified Public Accountant

Missouri State Medical Foundation Financial Statement - 1991

STATEMENT OF ASSETS AND FUND BALANCE - MODIFIED CASH BASIS DECEMBER 31, 1991

	1991
CURRENT ASSETS:	
Cash	$ 507,709
Investments	501,498
Loans receivable - current	220,000
Total Current Assets	1,229,207
LONG-TERM ASSETS	
Loans receivable - long term	1,108,570
Equipment - net	983
Total Long-Term Assets	1,109,553
TOTAL ASSETS	$ 2,338,760
FUND BALANCE	$ 2,338,760

STATEMENT OF REVENUES, EXPENSES AND CHANGE IN FUND BALANCE - MODIFIED CASH BASIS YEAR ENDED DECEMBER 31, 1991

	1991
REVENUES:	
Interest on student loans	$ 96,254
Other interest and dividends	67,579
Contributions	8,239
Gain on sales of securities	5,753
Total Revenues	177,825
EXPENSES:	
Administrative	10,000
Professional fees	21,133
Printing and meeting	3,546
Provision for loan losses	7,494
Miscellaneous	6,372
Total Expenses	48,545
EXCESS REVENUES OVER EXPENSES	129,280
FUND BALANCE - BEGINNING	2,209,480
FUND BALANCE - ENDING	$ 2,338,760

NOTE 1 - SIGNIFICANT ACCOUNTING POLICIES

Organization:

Missouri State Medical Foundation was organized in 1961 for the purpose of making low interest loans to medical students.

Basis of Accounting:

The accounts are maintained, and these financial statements are presented on a modified cash basis of accounting. Under that basis, revenues are recognized when received rather than when earned, and expenses are recognized when paid rather than when incurred. Also, depreciation of fixed assets are reported in the financial statements.

Investments:

Investments are stated at the lower of aggregate cost or market at the balance sheet date.

Loans Receivable:

Loans receivable are stated at face amount less an allowance for doubtful accounts of three percent of the loan balances at year end.

Equipment:

Equipment is stated at cost less accumulated depreciation computed using the straight line method over the estimated useful lives of the assets.

Income Taxes:

The Foundation is qualified as an exempt organization under Section 501(c)(3) of the Internal Revenue Code and is not subject to federal or state income tax.

NOTE 2 - CASH

Cash includes the following accounts:

	1991
Cash in bank	$ 5,575
Money Market - bank	478,718
Money Market - Prudential	23,416
Certificates of deposit	-0-
	$ 507,709

The deposits in the bank exceed FDIC insurance by $384,293. The bank has not pledged collateral securities to insure these accounts.

NOTE 3 - INVESTMENTS

At December 31, 1991, the Foundation's portfolio of marketable securities consisted of securities with a cost of $501,497 and market value of $526,329. Gross unrealized gains on all marketable securities were $66,627, and gross unrealized losses were $2,352 for the year. Net realized gains included in revenues was $5,753. The costs of securities sold were determined on a specific share basis.

NOTE 4 - LOANS RECEIVABLE

The following is a summary of the transactions affecting the loans receivable account for the year:

Loans receivable 1-1-91	$ 1,206,153
New loans	372,280
Principal repayments	(214,585)
Loans written off	(2,589)
Loans receivable 12-31-91	$ 1,361,259
Allowance for doubtful accounts 1-1-91	$ 36,185
Current year provision	7,242
Loans written off	(2,589)
Allowance for doubtful accounts 12-31-91	$ 40,838

NOTE 5 - EQUIPMENT

Equipment is presented net of accumulated depreciation of $6,379. Depreciation expense was $89 for 1991.

NOTE 6 - RELATED PARTY TRANSACTIONS

The Foundation is assessed by the Missouri State Medical Association an annual fee for administrative cost. For the year ended December 31, 1991, the Foundation paid the Association $10,000 for administrative costs. The Foundation also reimburses the Missouri State Medical Association for expenses paid by the Association on behalf of the Foundation. This reimbursement for the year ended December 31, 1991 was $3,546 for printing and meeting expenses.

BREAST CONSERVATION THERAPY
The St. Luke's Hospital Experience

Delia M. Garcia, M.D., and Karen J. Halverson, M.D.
St. Louis, Missouri

Between 1979 and 1987, 170 patient with stages 0, I and II breast cancer were treated with breast conservation therapy. Twenty-eight women (16%) had intraductal carcinomas, 110 (65%) stage I disease and 32 (19%) had stage II breast cancers. Seventy-five percent of the patients received no adjuvant systemic treatment, whereas 20% received adjuvant chemotherapy and 5% were given Tamoxifen. All patients received radiation therapy to the breast after lumpectomy and, when appropriate, axillary dissection. Twelve patients (7%) recurred within the treated breast, whereas two patients (1%) recurred in regional lymph nodes. Fourteen women (8%) developed distant metastases and seven women (4%) developed contralateral breast cancer. The actuarial 5 year disease-free survival was 92% for the patients with intraductal carcinoma, 90% for T1 and 65% for T2 patients. Overall actuarial survival was 100%, 96% and 87%, respectively. The St. Luke's Hospital results are comparable to those reported in the literature.

We conclude that breast conservation therapy, including irradiation, is an alternative to modified radical mastectomy and that this option should be thoroughly discussed with the patient.

Breast-conserving surgery or lumpectomy -- whereby the gross tumor mass is removed with a varying margin of surrounding normal tissue -- followed by breast irradiation gives results comparable to those of mastectomy in local tumor control and survival.

Breast conservation therapy has been practiced extensively in Europe and Canada since the mid 1950s,[1-8] but this method of treatment did not become popular in the United States until the past 10 years. The Harvard Radiation Joint Center in Boston was the first center in the United States to use breast conservation therapy to treat a substantial number of patients.[9,10] The treatment is now available in most areas, yet the majority of American women undergo mastectomy for the treatment of breast cancer.

We report a group of patients with newly diagnosed, previously untreated breast cancer cared for at a community hospital by a small group of physicians. Patients received uniform radiation therapy primarily from the senior author.

Materials and Methods

Between 1979 and 1987, 170 women with stages 0, I, and II carcinoma of the breast were treated with breast conservation therapy at St. Luke's Hospital, a general community hospital in St. Louis.

The women ranged in age from 25 to 89 years, with a median age of 55. Figure 1 shows the number of patients referred for treatment. Many of the first patients treated with breast conservation surgery plus radiation were elderly women who were deemed medically inoperable, but the treatment gained popularity after publication of the initial results of the National Surgical Adjuvant Breast Project (NSABP) B-06.[11]

The majority of the patients were referred for treatment between 1985 and 1987. Approximately 50 new patients are now treated with breast conservation therapy annually, representing one-third of the breast cancer patients whose diagnosis is made at St. Luke's Hospital each year.

Four patients were black and 166 patients were white, reflecting referral patterns at this institution. Forty-two percent of the women were premenopausal. The preoperative mammogram was interpreted as suspicious or positive in 86% of cases, and 11% had mammograms interpreted as negative. Three percent of the patients did not have a prebiopsy mammogram.

Twenty-eight of the patients (16%) had stage 0 or intraductal carcinomas, 110 (65%) had stage I disease and 32 (19%) had stage II breast cancer.

Wide local excision (lumpectomy) was carried out in all patients and axillary dissection (usually levels I and II) in four (14%) of the patients with intraductal tumors, 88 of 110 (80%) of the patients with T1, and 25 of 32 patients (78%) with T2 tumors. Because of suspected or documented tumor at the margins of the initial excision specimen, re-excision was performed in six (21%) of the

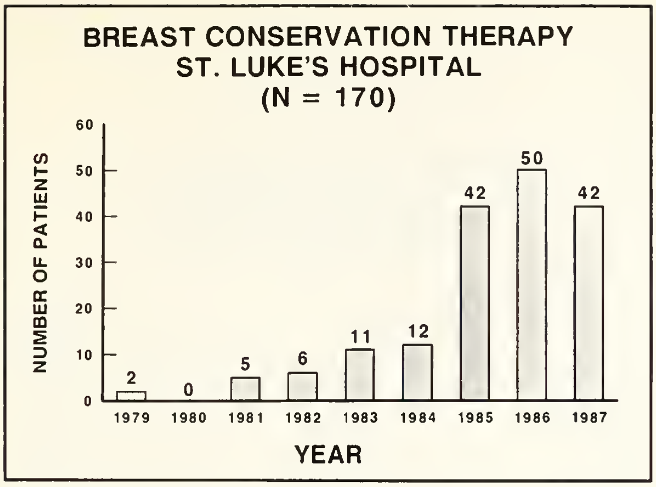


FIGURE 1: Number of patients referred to St. Luke's Hospital for treatment by year.

patients with intraductal, in 32 (29%) with T1 and in 12 (38%) with T2 tumors.

Radiation Therapy Techniques

Patients received radiation to the breast in tumor doses ranging from 4500 cGy in 25 fractions to 5040 cGy in 28 fractions with 6 MV photons. Breast irradiation was followed by a boost to the tumor excision site of an additional 1000 to 2000 cGy with electron beams in 153 patients. Four patients received a boost via an interstitial implant at the tumor excision site (2000 cGy over 48 hours). Eight patients received no boost.

Forty-two patients (25%) also received radiation to the regional nodes, supraclavicular and axillary, and received tumor doses ranging from 4600 cGy in 23 fractions to 5000 cGy in 30 fractions.

Presently, most patients receive irradiation only to the breast, but selected patients supraclavicular and axillary lymph nodes are treated to maximize local-regional tumor control. These patients have more than four positive axillary lymph nodes or extension of tumor outside the capsule of the lymph node or nodes.

Seventy-five percent of patients received no adjuvant systemic therapy. Five percent received tamoxifen. Of the remaining 20%, 31 patients received adjuvant chemotherapy with cytoxan, methotrexate and 5-Fluorouracil, and three received adriamycin based chemotherapy.

All patients were observed until their death or for at least 3 years.

Results

In the 28 patients with intraductal carcinoma, two (7%) developed a recurrence in the breast; no nodal or distant metastases were noted. Figure 2 shows the anatomical location of tumor recurrences by stage in the patients with T1 and T2 tumors. Of the 12 patients (11 of whom had received electron boosts) whose cancer recurred in the treated breast, 11 patients underwent mastectomy, and one patient had a wide local excision.

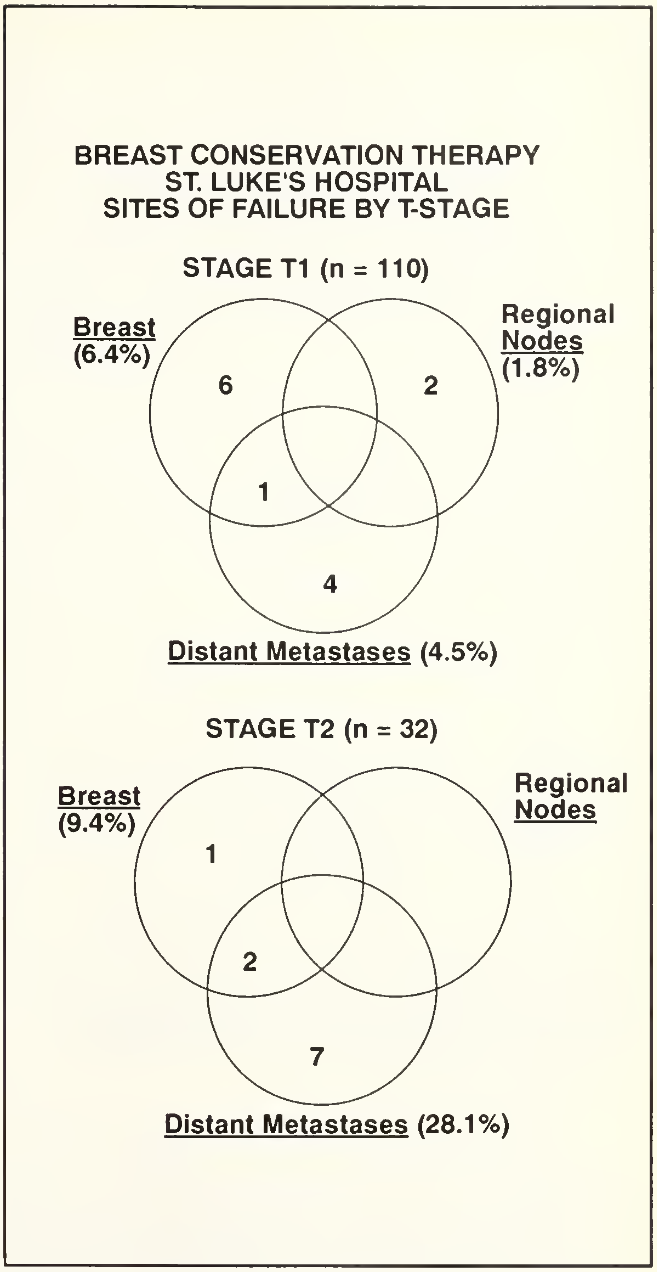
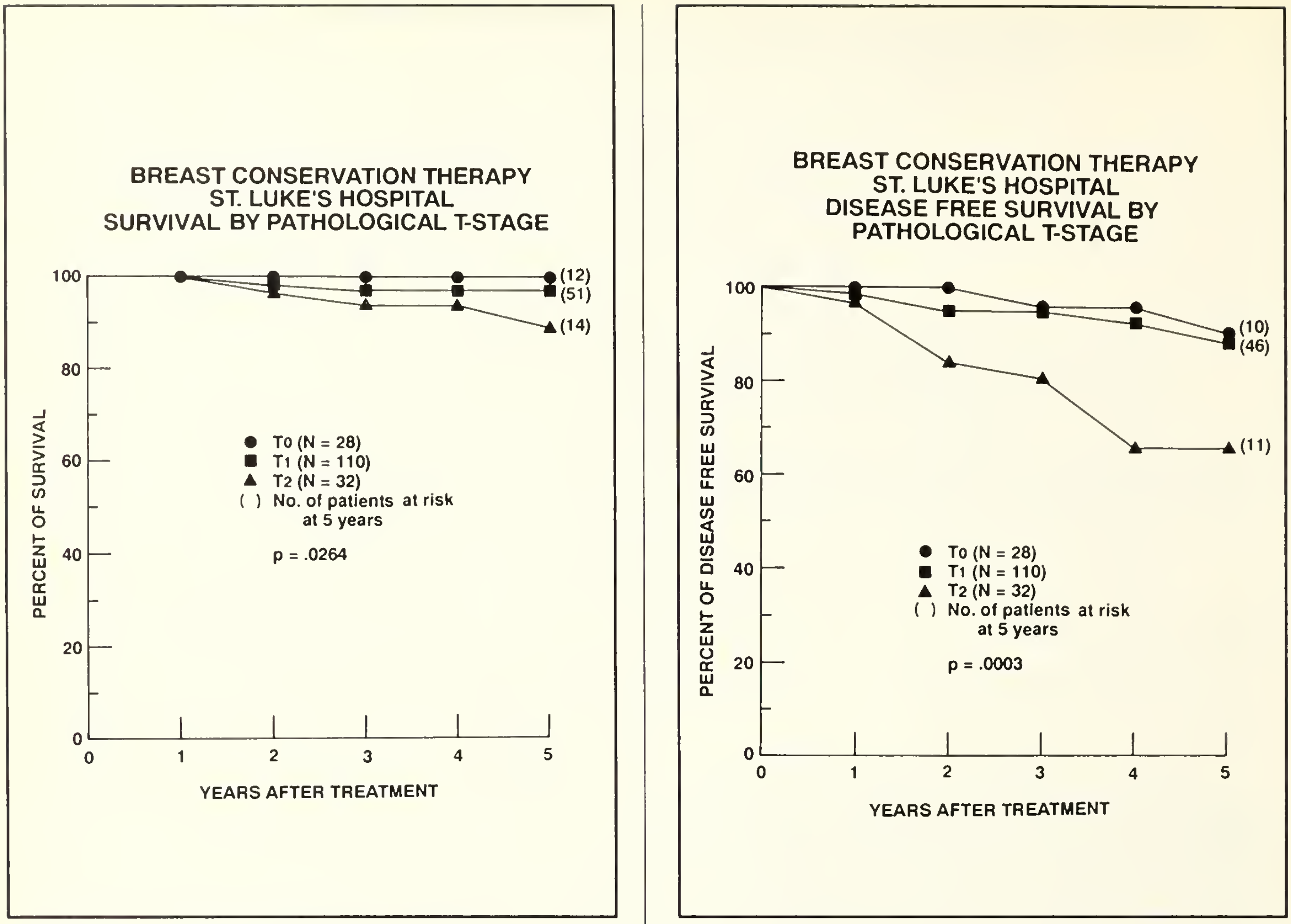


FIGURE 2: Anatomical site of tumor recurrences by stage in 170 women treated with breast conservation therapy.

Three patients who had recurrence in the breast received chemotherapy, and one patient was treated with tamoxifen. Two women who received systemic therapy had concurrent distant metastases; both patients



FIGURES 3A Survival AND 3B Disease-free survival for patients with T1 and T2 tumors treated with breast conserving therapy.

eventually died as a result of distant disease.

One patient who had a recurrence of cancer in the treated breast developed metastases 16 months after the recurrence. She is alive, but with disease. The remaining nine patients with disease recurrence in the ipsilateral breast are alive with no evidence of tumor.

The woman who had a recurrence of disease in the axilla was clinical stage T1N0 at diagnosis, as she did not have an axillary dissection. She received irradiation to both the breast and regional nodes. Currently, she is alive and free of disease after excisional biopsy of an axillary nodal recurrence.

The patient who had a recurrence in the supraclavicular lymph nodes was pathological stage T1N0 at diagnosis and received irradiation to the breast only. She is alive, but has known local tumor and distant metastases. In addition, seven women (4%) developed a new tumor in the contralateral breast.

Cosmetic results were judged to be "good" to "excellent" in 83%, and "fair" in 12% of the patients (Table 1).

At the time of this report, 93% of the patients were alive, and 96% of these patients were free of disease (Fig 3). Four percent were living, with known tumor. Twelve patients (7%) had died. Of the patients who died, 67% had metastatic breast cancer, 25% died of intercurrent disease, and 8% died of a second primary cancer.

Table 2 shows the complications in our treatment population, breast edema being the most frequent (7%), followed by fibrosis (5%).

Discussion

Between April 1976 and January 1984, the NSABP conducted a prospective randomized trial (protocol B-06) involving 1,843 women with stages I and II breast cancer.[11,12] Eligible women were randomized to three treatment arms: 1) total mastectomy; 2) lumpectomy

Table 1
BREAST CONSERVATION THERAPY
ST. LUKE'S HOSPITAL
COSMESIS AT LAST EVALUATION

	Pathological Stage			
COSMESIS	T-O	T-1	T-2	TOTAL
Excellent	26 (93%)	93(85%)	22(69%)	141(83%)
Fair	2 (7%)	13(12%)	5(16%)	20(12%)
Poor	0 (0%)	4(4%)	5(16%)	9(5%)
	28 (16%)	110(65%)	32(19%)	170

only; and 3) lumpectomy and breast irradiation. Patients with positive axillary lymph nodes received adjuvant chemotherapy. The eight-year results of the trial[12] showed no significant differences in disease-free survival and overall survival, regardless of treatment. However, women treated with lumpectomy- only had a significantly higher incidence of tumor recurrence in the ipsilateral breast compared with those who received breast irradiation. Table 3 shows the local recurrence rates according to treatment and status of the axillary lymph nodes. Patients who received both breast irradiation and chemotherapy had the lowest incidence of recurrence in the breast, implying a synergistic effect between the radiation and chemotherapy.

The NSABP reports have been influential in increasing the number of patients referred for breast conservation therapy. With more women choosing lumpectomy plus radiation in lieu of mastectomy, it is important for each center treating such patients to carefully review its own clinical results. Our results are comparable to the NSABP B-06 results, both in survival and in local tumor recurrence rates.

Women who undergo breast conservation therapy tolerate the treatment extremely well. In fact, the recovery time following lumpectomy may be shorter than that after mastectomy because of the less extensive operation. Radiation therapy is usually initiated two weeks after operation, once the wound has healed adequately. Radiation treatment may be delayed for chemotherapy, but patients who are to have adjuvant therapy with cytoxan, methotrexate, 5-FU, or tamoxifen can receive concomitant radiation and systemic therapy. If adriamycin-based chemotherapy is recommended, radiation therapy is postponed until chemotherapy is completed. Alternatively, the adriamycin may be withdrawn after two or three cycles, the radiation treatment completed, then adriamycin reinstituted into the remaining cycles of chemotherapy.

Table 2
BREAST CONSERVATION THERAPY
ST. LUKE'S HOSPITAL
1979 - 1987
N=170

COMPLICATION	Number of Patients (%)
BREAST EDEMA	12 (7)
BREAST FIBROSIS	8(5)
ARM EDEMA	4(2)
SOFT TISSUE NECROSIS	2(1)
RIB FRACTURE	1
PNEUMONITIS	1
TOTAL	28(16)

Table 3
NSABP B-06
RECURRENCE RATES IN THE IPSILATERAL BREAST

	Lumpectomy Only	**Lumpectomy + RT**
Negative Nodes	37%	12%
Positive Nodes*	43%	6%

NEJM, 1989

* These patients also received chemotherapy

The main side effect from breast irradiation is erythema and dry desquamation, which usually begins three weeks into the treatment and resolves within two to four weeks of completing irradiation. Patients may experience fatigue and, if the lymph nodes are irradiated, a mild sore throat. Complications of breast irradiation are relatively rare but include symptomatic radiation pneumonitis. This occurs when 1 to 2 cm of underlying lung has been exposed to radiation in order to reach the deepest portion of the breast. Also, if the supraclavicular and axillary lymph nodes are irradiated, the apex of the lung is in the treatment field and pneumonitis is more likely. Overall, symptomatic pneumonitis is seen in approximately 5% of patients and is more common if the regional lymphatics are irradiated.

Pneumonitis is generally self-limited, but it may take several weeks or even a few months to resolve completely. In some patients, steroids and anti-inflammatory medications provide symptomatic relief. Edema of the breast and upper extremity occur in approximately 5% of the patients and is partially attributable to axillary dissection. Two to three percent of women develop spontaneous rib fractures months to years after the irradiation, but the fractures heal on their own.

Breast conservation therapy raises quality of life issues. Overall, 83% of our patients had a good to excellent cosmetic result. After treatment, every patient was questioned and all stated they would opt for breast conservation therapy again.

Summary

Breast conservation therapy has become an increasingly popular form of treatment for patients with *in situ* and stages I and II carcinoma of the breast. The survival rates and local control of the tumor are equal to that of mastectomy, with the major difference being a better cosmetic result. St. Luke's Hospital's results with breast conservation therapy are comparable to the reported results in the literature. The treatment is well tolerated with an acceptable incidence of side effects and complications and offers an alternative to women who do not desire mastectomy. Close surveillance after treatment is essential, since early detection of disease recurrences allows the best chance for cure.

References

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Acknowledgement: The authors would like to thank Dr. Carlos Perez for his valuable input in the review of this manuscript.

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The Neurological Aspects of Acquired Immune Deficiency Syndrome (AIDS)-A Clinical Review

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University of Missouri-Columbia

Ten years after it was diagnosed, AIDS is estimated to have affected 10 million people worldwide and about 2-3 million people in the United States. Initially considered to be a disease of coasts (New York, California, Florida), AIDS is making inroad to mid-America as well. It involves the nervous system in a variety of ways. These include direct involvement by the virus, secondary opportunistic infections, and tumors. The spectrum of nervous system involvement is the subject of our review.

Since recognition of the syndrome in 1981, 167,803 cases of Acquired Immunodeficiency Syndrome (AIDS) have been reported in the United States.[1] Of these, 106,361 patients (63.4%) have died. It is estimated that by 1991, there will be 350,000 cases of AIDS Related Complex (ARC) and about 2-3 million asymptomatic cases in the United States. About 10 million asymptomatic cases are expected to be in the world at this time. As recent trends indicate, AIDS has made inroads into mid-America.

Since 1982, 2,019 cases of AIDS have been reported in Missouri, of which 1,073 people (53%) have died.[1] Among adults affected with the disease in the United States, 73% are homosexual or bisexual men. Seventeen percent of the cases are in intravenous drug abusers. Haitian immigrants to the United States constitute about 7% of cases of AIDS. The remaining 7% are those who contracted the disease via blood transfusions or heterosexual contact.[2]

Etiology

AIDS is caused by the Human Immunodeficiency Virus (HIV), a lymphotropic human retrovirus. It selectively infects the helper subset of human T-lymphocytes called CD-4 cells. This leads to the reversal of the normal CD-4 to CD-8 ratio (helper to suppressor ratio).

Pathogenesis

It is postulated that HIV enters the brain via infected T-helper lymphocytes or monocyte macrophages. Neurons do not appear to be directly infected by HIV. Brain macrophages probably act as reservoirs for the virus. The virus is not cytopathic to the brain.[3] Cytokines released by infected macrophages impair cellular function. These soluble factors cause leukoencephalopathy by binding CD-4 receptors in the hippocampus and amygdala. The rate of decline in the CD-4 lymphocytes may be a predictive factor of subtle neuropsychological abnormalities in patients with HIV infection.[4].

Clinical Features

Patients with AIDS may initially come to a physician with a variety of disease complaints. The most common initial diagnoses include

- *Pneumocystis carnii* Pneumonia (50%)
- Kaposi Sarcoma (25%)
- *Pneumocystis carnii* Pneumonia *and* Kaposi sarcoma (10%)

The remaining 15% may have other disease entities, including infection with candida, herpes, or mycobacteria, and central nervous system disorders.[5]

Neurologic Features

Neurological dysfunction is a frequent complication of AIDS. Approximately 60% of patients with AIDS have neurological symptoms. From 80% to 90% of patients are found to have neuropathological abnormalities at autopsy.[6] Figure 1 indicates various neurological complications of AIDS and ARC.

CENTRAL NERVOUS SYSTEM

Brain Involvement

Acute HIV Meningoencephalitis

This may be the earliest manifestation of AIDS. Within days or weeks after exposure to the virus, the patient may experience an acute febrile syndrome[7] consisting of fever, headache, and meningsmus.

The CSF shows mononuclear pleocytosis, normal or mildly decreased glucose, normal or mildly increased protein and culture for microorganisms is negative. The CT scan is normal. Any EEG changes reflect the level of consciousness and are non-focal. Most patients recover from the episode, although a few may develop chronic meningitis. The HIV infection, of course, remains.

AIDS Dementia Complex

This is the most common neurological manifestation. Between 50% to 80% patients with HIV infection are affected with AIDS dementia complex. The first signs of mental dysfunction may be missed due to the presence of other life endangering complications. The patient may have progressive apathy toward work, social and recreational activity. There may be mental slowing, intellectual impairment, and behavioral changes that may later progress to severe dementia.[8] Accompanying dementia, there may be other features such as abulia, mild unilateral or bilateral pyramidal signs, grasp or snout reflex, gait apraxia, dysdiadochokinesis, tremors, and myoclonus. In 50% of patients with AIDS dementia complex, paraparesis may occur secondary to the vacuolar myelopathy.[6] The CSF shows mild inflammatory changes and HIV can be found in CSF in 50% of cases. The CT scan shows cortical atrophy, dilated ventricles and white matter rarefaction. Electroencephalography is rarely helpful in distinguishing AIDS dementia complex from other dementing disorders.[6] Pathologically, diffuse pallor in white matter is noted. In some cases, it is accompanied by scanty perivascular infiltrates of lymphocytes and brown pigment macrophages. In advanced cases, distinct foamy macrophages and multinucleated cells associated with multifocal rarefaction of white matter are seen.[9]

Chronic Meningitis

The patient may present as having chronic headaches and meningeal signs. On occasion, cranial nerves 5, 7 and 9 may be involved. The CSF may show chronic inflammatory changes. Although HIV may be recovered from the CSF in patients with aseptic meningitis, there may be no correlation between recovering of virus and the clinical severity of neurological disease.[3]

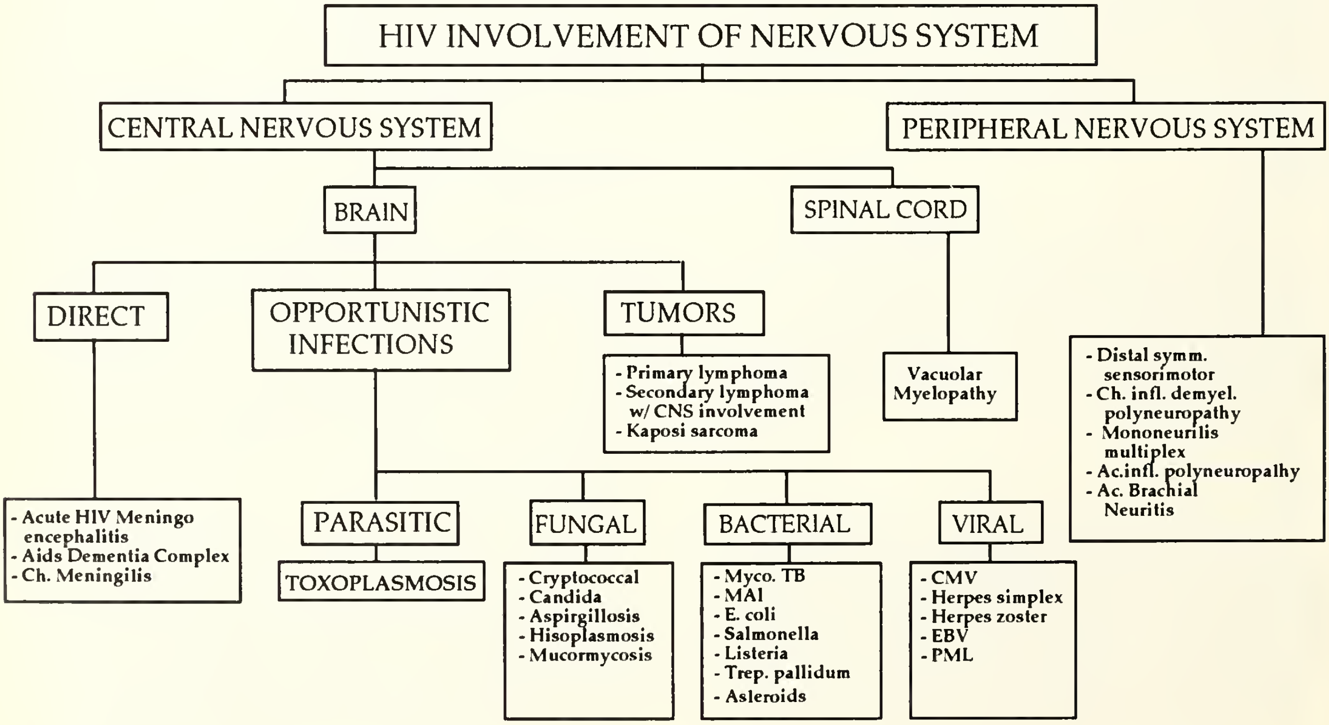
Opportunistic Infections of the Brain

Parasitic

Toxoplasmosis

Toxoplasmosis is the most common cause of brain abscess in patients with HIV. This infection is caused by *Toxoplasma gondii*, an obligatory intracellular

Fig. 1 Various Neurological Complications of Acquired Immune Deficiency Syndrome and AIDS Related Complex.


HIV INVOLVEMENT OF NERVOUS SYSTEM
CENTRAL NERVOUS SYSTEM
PERIPHERAL NERVOUS SYSTEM
BRAIN
SPINAL CORD
DIRECT
OPPORTUNISTIC INFECTIONS
TUMORS
- Primary lymphoma
- Secondary lymphoma w/ CNS involvement
- Kaposi sarcoma
Vacuolar Myelopathy
- Distal symm. sensorimotor
- Ch. infl. demyel. polyneuropathy
- Mononeurilis multiplex
- Ac.infl. polyneuropalhy
- Ac. Brachial Neuritis
- Acute HIV Meningo encephalitis
- Aids Dementia Complex
- Ch. Meningilis
PARASITIC
TOXOPLASMOSIS
FUNGAL
- Cryptococcal
- Candida
- Aspirgillosis
- Hisoplasmosis
- Mucormycosis
BACTERIAL
- Myco. TB
- MAI
- E. coli
- Salmonella
- Listeria
- Trep. pallidum
- Asleroids
VIRAL
- CMV
- Herpes simplex
- Herpes zoster
- EBV
- PML

parasite that has a worldwide distribution and appears to be ubiquitous in the United States, with a seroprevalance of 40% to 50%. Except for the disproportionate number of cases in Haitians, toxoplasmosis is evenly distributed among different risk groups. The higher incidence of cases in Haitians reflects the higher rate of seropositivity in adults from underdeveloped areas of the world.[10]

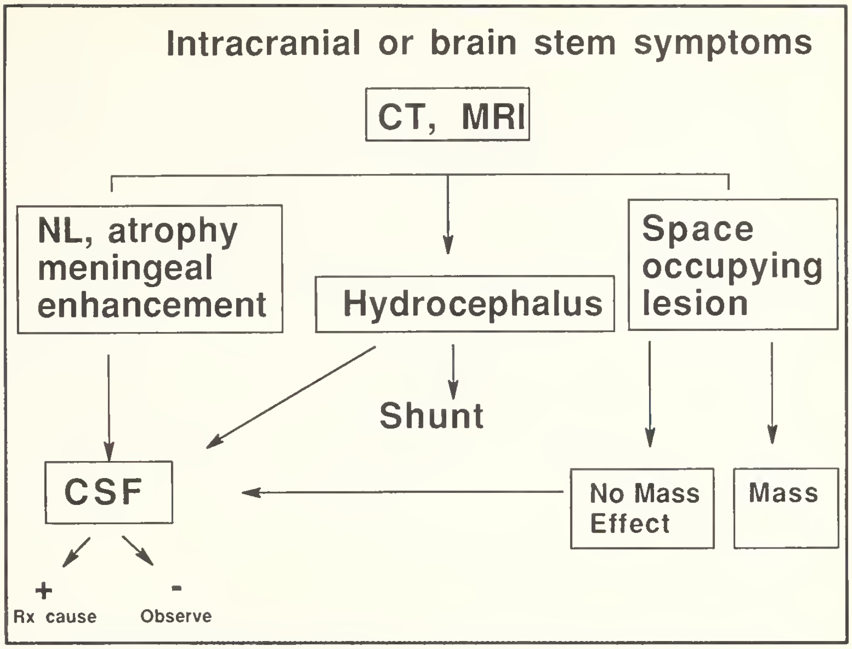

Intracranial or brain stem symptoms
CT, MRI
NL, atrophy meningeal enhancement
Hydrocephalus
Space occupying lesion
Shunt
CSF
No Mass Effect
Mass
+
-
Rx cause
Observe

Fig. 2: Algorithm for the evaluation and treatment of intracerebral or brain stem symptoms in patients infected with HIV.

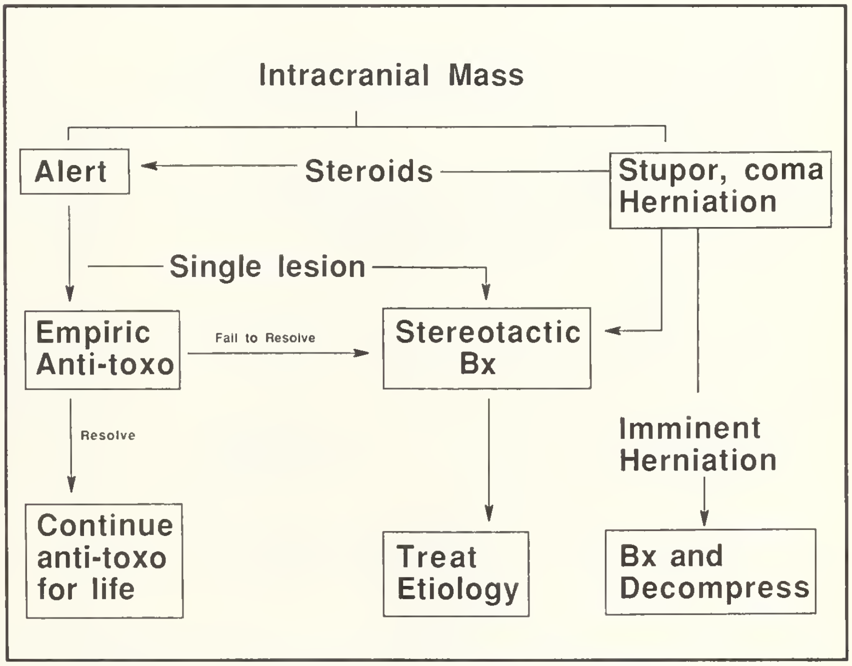

Intracranial Mass
Alert
Steroids
Stupor, coma Herniation
Single lesion
Empiric Anti-toxo
Fail to Resolve
Stereotactic Bx
Resolve
Imminent Herniation
Continue anti-toxo for life
Treat Etiology
Bx and Decompress

Fig. 3: Algorithm for evaluation and treatment of intracranial map lesions in patients with HIV.

The latent infection leaves cysts in the muscles and brain. Because of immunosuppression due to AIDS, patients with the syndrome experience endogenous reactivation of cerebral toxoplasmosis. Clinically, the patient may have a variety of focal neurological deficits, including hemiparesis, hemianopsia, aphasia, seizures, or mental impairment such as confusion, apathy, or subeuphoric state.

The Sabin Feldman dye test and IgG indirect fluorescent antibody (IFA) tests seem to be useful in the diagnosis of toxoplasmosis. A titer in excess of 1:1024 with Sabin Feldman dye test, and greater than 1:512 with IFA, indicate active disease. Elevated titers of IgM-IFA usually indicate infection within the previous three months.[3] The CSF may show nonspecific inflammatory changes, and CT scan usually shows large, multifocal lesions with ringlike enhancement using contrast. The lesions are multifocal with predilection for the gray matter. The EEG may show generalized changes in basal activity plus focal abnormalities.

In early stages, lesions of toxoplasmosis are soft, edematous, dusky and poorly defined. As the infection progresses, necrosis and edema develop around the lesion. Microscopically, the lesions are characterized by small microglial aggregates, inflammation, and reactive gliosis. The definitive diagnosis is made by demonstration of organisms in the parenchyma around a central area of necrosis. Brain biopsy is not performed in all patients suspected to have toxoplasmosis, but is indicated for those with solitary lesions, a history of sulfonamide intolerance, and clinical deterioration despite an adequate trial of empirical toxoplasmosis therapy. When biopsy is attempted, the material must be obtained from the periphery of lesion because it is often difficult to find organisms in the central necrotic portion of active lesions.[3]

A patient suspected to have toxoplasmosis should receive treatment with 100 mg pyrimethamine, a dihydrofolate reductase inhibitor, along with 4 g of sulfadiazine. After that, the patient receives daily doses of 50 mg pyrimethamine and 500 mg sulfadiazine. Folinic acid is also given to prevent hematoxicity. If the patient is allergic to sulfa,

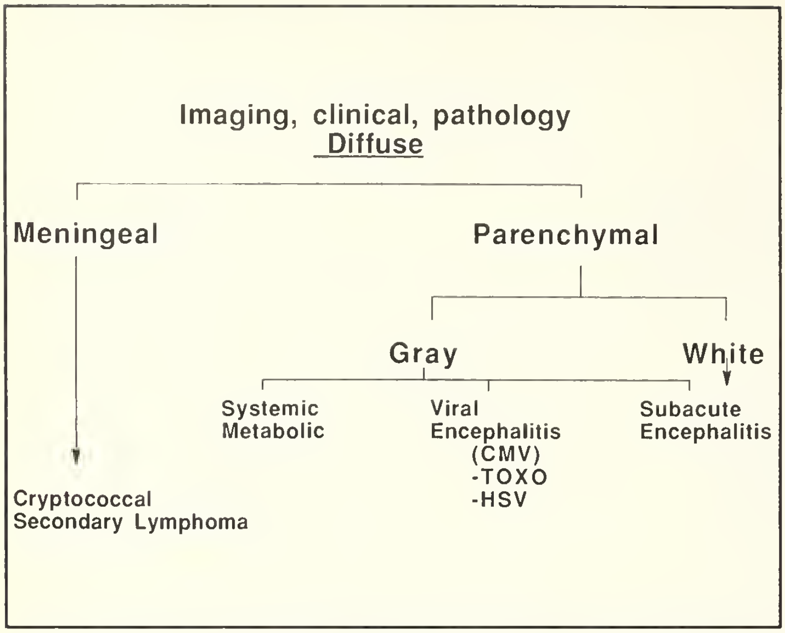


Fig. 4: Diagram showing simplified approach to the differential diagnosis of opportunistic infections and neoplasms in patients with AIDS.

Algorithms reproduced with permission of Mark L. Rosenblum, M.D., author of "AIDS and the Nervous System" and Surl Nielson, M.D., author of Neuropathology of AIDS in "AIDS and the Nervous System."

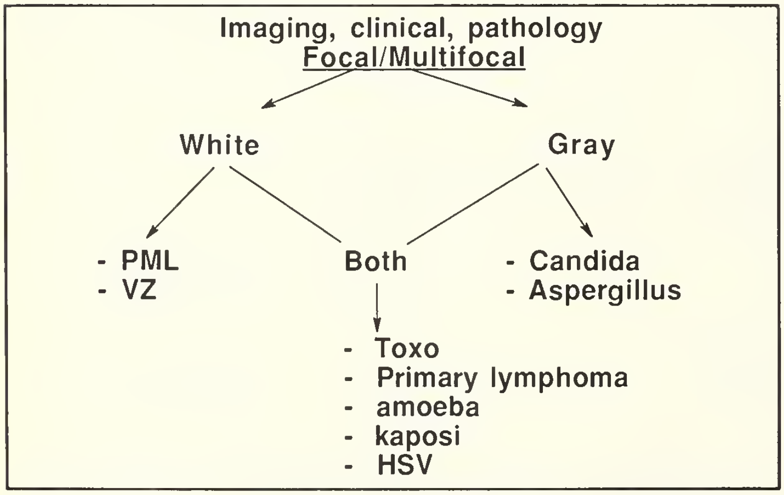


Fig. 5: Diagram showing simplified approach to the differential diagnosis of opportunistic infections and neoplasms in patients with AIDS.

pyrimethamine alone may be tried. Many believe that the treatment must be given for the rest of the patient's life. If there is no improvement in the patient's condition, biopsy may be needed to rule out other disease processes such as infection with *Mycobacterium avium* intracellulare, herpes simplex, or progressive multifocal leukoencephalopathy, Kaposi sarcoma, and lymphoma.

Fungal Infections

Cryptococcosis

Cryptococcus neoformans is an infrequently found but virulent pathogen among patients with AIDS.[11] It is commonly found in soil, particularly where there are pigeons, and enters the body by respiratory inhalation of aerosolized organisms. Pulmonary disease is frequently asymptomatic, but hematological dissemination to other organs can occur.

For reasons which remain unclear, the central nervous system is particularly vulnerable to cryptococcal infection and is the most common site of clinically apparent cryptococcal infection in AIDS. The prevalence of cryptococcus in patients with AIDS ranges from approximately 2% to 9%, with a somewhat higher prevalence among blacks and intravenous drug abusers. The development of cryptococcosis generally indicates profound immunosuppression. *Cryptococcus neoformans* is also the most common cause of meningitis in patients with HIV.[12]

Patients have the insidious onset of headache, fever, malaise, and altered mental status. The CSF usually shows mononuclear pleocytosis, depressed glucose and moderately elevated protein. Cryptococcal antigen titers are elevated and India ink preparation will show the organisms. When cryptococcal infection is disseminated, blood cultures are positive and serum titers elevated.[13] Findings on CT scan may be normal, but focal lesions - cryptococcomas - have been reported. Changes on the EEG are consistent with mental status changes (progressive diffuse slowing). The subarachnoid space is distended by a thick gelatinous exudate. Microscopic examination shows numerous yeast forms, and expansion of the perivascular space with resultant formation of cryptococcomas. Amphotericin-B 16 mg/kg body weight per day is given for six weeks. During successful treatment, both serum and CSF cryptococcal antigen levels are expected to fall and the fungal culture will become negative. Flucytosine, the other drug used for cryptococcosis, is rarely used in AIDS patients because of its potential to cause diarrhea and myelosuppression. Fluconazole, another oral antifungal, appears to be far less toxic than amphotericin B and crosses the blood-brain barrier. It has shown promise for primary therapy and as a maintenance agent.[14]

Candidiasis

Despite its being a common mucosal infection, candidal infection in the CNS is rare in patients with AIDS. Brain abscess may be present and focal findings are found depending upon the site of the lesion. The lesion may be demonstrated by contrast enhanced CT scan. Treatment is with amphotericin-B.

Other Mycoses

Aspergillosis is a rare infection that occurs generally in the terminal stages of AIDS. It may manifest as meningoencephalitis or intracranial abscess.

Coccidiodomycosis occurs only in endemic areas of the southwestern United States. It produces basilar granulomatous meningitis, which may cause vasculitis and prominent thickening of the meninges. This may result in cerebral infarction. For definitive diagnosis, CSF complement fixation titers may be needed. The CT scan shows mild atrophy and no parenchymal lesions.

Histoplasmosis is another CNS pathogen prevalent in the Mississippi valley and surrounding areas. It is associated with multiple foci of parenchymal necrosis, particularly in the gray matter.

Mucormycosis produces angiotropic lesions and microabscesses.

Bacterial Infections

By virtue of its interrelationship with cell-mediated immunity, the humoral immune system is also impaired in AIDS, and a variety of bacterial infections are found.

Mycobacterium tuberculosis

This infection is particularly prevalent in Haitians and intravenous drug abusers. Both focal parenchymal and tubercular meningitis has been reported. Newly diagnosed TB is an indication to test for HIV. While pulmonary infection with *Mycobacterium tuberculosis* is common, the PPD may be negative due to anergy.[15]

Mycobacterium Avium Intracellulare (MAI)

This has been a major cause of disseminated mycobacterial disease in patients with AIDS. Treatment of MAI is difficult because the organism is usually resistant to most antitubercular treatment. Even in non-AIDS patients with *Mycobacterium avium intracellular* infection, the cure rate after treatment with six or seven antitubercular agents is only 30%. Isoniazid, Rifampin, Ritabulin, pyrazinamide, and clofazamine may be tried.[16]

Other bacteria that infect the CNS of patients with AIDS are *Staphylococcus aureus, Streptococcus pneumoniae,* Nocardia, Listeria, Salmonella, and asteroides. Neurosyphilis is relatively common in asymptomatic HIV patients, and should be treated.[17]

Viral Infections

Cytomegalovirus (CMV)

In patients with AIDS, cytomegalovirus is the most common cause of blindness. The virus may also cause encephalitis, polyradiculitis, and retinitis and may be a cofactor in the activation of HIV in the brain, thus contributing to the development of HIV encephalopathy. Serological studies do not reliably distinguish patients with symptomatic disease from those with latent infections. The CSF changes are nonspecific, although inclusion bearing cells have been reported. An MRI scan of the brain may show generalized atrophy and irregular periventricular enhancement. Macroscopically, the cortical surface of brain may show mild atrophy. The ventricular surface may bear evidence of infection of the ependyma and frank hemorrhagic ventriculitis. Microscopically, CMV encephalitis may show microglial nodules, inclusion bearing glial cells, focal parenchymal necrosis, necrotizing ventriculoencephalitis and necrotizing radiculomyelitis.[12] CMV infection is treated with ganciclovir, but the benefit is transient.

Herpes Simplex

Type I Type I is acquired non-venereally during childhood and remains latent in the olfactory or trigeminal ganglia. The virus is reactivated during immunosuppression. The patient may have acute headache, behavioral changes, focal seizures and coma. The CSF may show increased pressure, mononuclear pleocytosis, decreased glucose and elevated proteins. The CT scan may show areas of hypodensities in temporal and frontal regions. The EEG may show periodic lateralized epileptiform discharges.

Type II Herpes simplex type II is acquired during sexual exposure. During immunosuppression, the virus reactivates from the sacral ganglia. In a study done by Corey, 36% of women and 13% of men who were immunocompromised developed meningitis from herpes simplex virus type II. The incidence of herpes simplex I seems to be decreasing, while type II is increasing in this country.[13] Treatment of herpes simplex is by acyclovir and the dosage is 30 mg per kg per day for 10 days.

Varicella Zoster (VZV)

There is a strong association between herpes zoster infection and defects in cell-mediated immunity. However, in AIDS patients, who have defects in overall immunity, there are few neurological complications with varicella zoster. Evidence that VZV is directly responsible for CNS disorders in HIV is mainly circumstantial. Microscopically, Cowdry A intranuclear inclusions and herpes A virus-like particles have been identified.[19] Infection with VZV is treated with acyclovir in a dosage of 10 mg per kg per day. Relapse is common.

Epstein Barr Virus (EBV)

EBV infection is quite common in patients with AIDS and has been associated with a Burkitt-like lymphoma. Other manifestations include encephalitis, acute cerebellar syndrome, transverse myelitis, and ascending myelitis.[20]

Progressive Multifocal Leukoencephalopathy (PML)

Progressive multifocal leukoencephalopathy is an opportunistic infection of the CNS caused by a member of the JC virus family. Antibodies to JC virus are present in approximately 70% of individuals in the general population. Most cases of PML are reactivation of latent infection rather than new infection. After CMV, PML is the common virus infection in patients with AIDS. The patient may have the insidious onset of mental status changes and focal neurological deficit. The CT scan may show multiple asymmetrical low density lesions particularly in the subcortical white matter. Macroscopically, PML is characterized by multiple, randomly distributed areas of demyelination in the subcortical area. Smaller lesions may be translucent while larger lesions may be soft and cystic. Microscopically, lipid laden macrophages, bizarre giant astrocytes, and enlarged oligodendrocytes with basophilic and eosinophilic intranuclear inclusions are found. The biopsy may reveal the presence of JC virus, identified by dot hybridization. Although PML is fatal, there are reports of favorable response to cytosine arabinoside.[21]

Tumors

CNS Malignancies

There is a high overall incidence of malignancies in patients with AIDS. Five percent of patient with AIDS may have otherwise uncommon primary lymphoma.

Primary Lymphoma

This is a high grade tumor of B-cell origin. The patient may present with hemiparesis, aphasia, and convulsions. Increased intracranial pressure may cause headache and vomiting. Confusion, lethargy and memory loss may also be seen. CSF may show increased protein, decreased glucose, and mononuclear cells. Cytology for malignant cells may be negative. CT scan may show multiple focal lesions. the lesions are often contrast enhancing and have surrounding edema. EEG may show focal or generalized changes. Primary lymphoma is treated by 4,000 rads of radiation therapy over 3 weeks.[10]

Systemic Lymphoma

Systemic lymphoma may present as primary CNS tumor due to cerebral and meningeal involvement. CSF may show characteristic lymphoma cells. in patients with acute compressive myelopathy, CT scan and myelography may be needed. Intratretheal methotrexate and Arabinosyl cytosine is recommended. However, overall prognosis is poor.

Kaposi Sarcoma

It is the most common malignancy of the skin in patients with HIV. For unknown reasons, however, it is rare in CNS. Examination reveals focal findings depending upon the site of involvement. CT scan shows the site and extent of involvement. Palliative treatment is by radiation therapy.

Spinal Cord Involvement

At autopsy, 10-20% of AIDS patients are found to have vacuolar myelopathy. Usually seen in thoracic spinal cord, it involves posterior and lateral funiculi. The patient may present with paraparesis, spasticity, ataxia, leg weakness, painful burning sensation, paraesthesia, decreased proprioreception and incontinence. microscopically, vacuoles seem to represent periaxonal and intramyelinic edema.

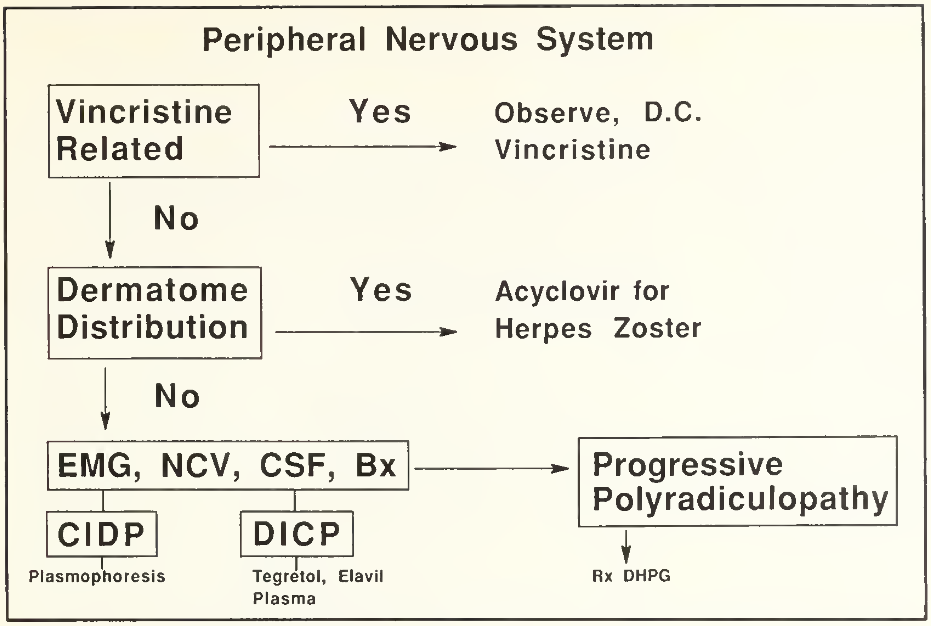


Fig. 6: Algorithm for the evaluation and treatment of peripheral nervous system in patients infected with HIV.

PERIPHERAL NERVOUS SYSTEM

Dysfunction of peripheral nervous system has been reported in 15-50% of patients with AIDS. However, it is grossly underdiagnosed due to the presence of severe, debilitating and life threatening conditions present in patients with AIDS. These patients are susceptible to a wide variety of peripheral nervous system complications. Possible etiologies include varicella zoster, herpes virus, nutritional, toxic or metabolic factors.

Diffuse Symmetrical Sensorimotor Neuropathy

This is an axonal neuropathy and is most common in the late stage of AIDS. The patient may present with painful dysaesthesia, and numbness. Thinning of skin, pedal edema, and marked weakness in distal muscles of lower extremities is noted. There is associated areflexia. Nerve conduction studies done after warming the distal limbs, show normal conduction velocity. EMG usually shows fibrillations and positive waves in the distal muscles of lower extremities. Large motor unit potentials are seen.[10] Treatment is with Tegretol or Amitriptyline. Rarely ankle foot orthosis may be needed.

Chronic Inflammatory Demyelinating Polyneuropathy

It occurs in early stages of AIDS. Presenting features include proximal weakness, mild sensory deficit, decreased vibrations and areflexia. nerve conduction studies show prolonged or absent F waves and prolonged distal motor latencies. Also, patchy marked slowing may be noticed and different findings may be obtained between proximal and distal segments of the same nerve. EMG may show normal spontaneous activity and motor unit potentials are of normal configuration. Treatment modalities include steroids, plasmapheresis and high doses of intravenous gammaglobulins.

Mononeuritis Multiplex

This is an axonal neuropathy. Multiple spinal and cranial nerves, specially trigeminal and facial nerves, may be involved. Peripheral nerve involvement may cause wrist and foot drop. Electrophysiological studies may show features consistent with axonal neuropathy with or without evidence of demyelination.

Acute Inflammatory Polyneuropathy

It is a demyelinating neuropathy. There is progressive motor and sensory deficit beginning at lumbar and sacral roots. There is an early impairment of bladder and rectal sphincter control. EMG may show reduced recruitment pattern in weak muscles.

DIAGNOSIS

The diagnosis of AIDS relies on presence of empirically defined secondary complications of underlying immune defect. A highly sensitive and readily available enzyme linked immunosorbent assay (ELISA) exists for detection of antibodies against HTLV-III. Western blot analysis which establishes the specificity of immunological reaction between antibodies and viral encoded proteins can be further used to confirm positive ELISA.

TREATMENT OF AIDS

The treatment is increasingly frustrating as there is rapid downhill course. Management of opportunistic infections has been discussed. But unless underlying immune system can be corrected, management is mainly palliative. AZT (Zidovudine) has an excellent "in vitro" activity against HIV. It has 100% inhibition of replication by cytopathic effect on HIV.[22] AZT is a structural analogue of Thymidine and acts as a competitive inhibitor of the reverse transcriptase activity of HIV. The drug appears to be virustatic and crosses blood brain barrier.[23] AZT has been shown to decrease mortality and the frequency and severity of opportunistic infections in patients with AIDS or ARC.[23]

Recently FDA has approved Dideoxyinosine as a treatment modality. There is a prominent side effect of peripheral neuropathy and pancreatitis. Didanosine is another drug undergoing trial, which also has side effects of peripheral neuropathy. Interferon, Interleukins, Suramins, HPA-23, have been unsuccessfully tried in the past. No immunization against HIV is available at this time.

Practical Approach to HIV Patients with Neurological Complications

A thorough history and a complete physical exam including neuropsychological evaluation should be done. Lumbar puncture should be done to check for cryptococcal infection. CT scan with contrast enhancement is done to differentiate between various opportunistic infections and Aids Dementia Complex. MRI is preferred if available. Biopsy is needed for definite diagnosis. However, it carries risks of hemorrhage or being false negative or difficulty to approach the lesion. Electromyography and nerve conduction studies may help in diagnosing nerve muscle problems. The above mentioned management algorithms may be helpful in differentiating various clinical entities seen in patients with AIDS and thereby help in treatment of these patients. Nevertheless, it is an uphill battle.

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ACKNOWLEDGMENT: The authors would like to thank Miss Michelle Dettmer for transcription of this manuscript.

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■ **Lawrence W. Feltz, M.D.**, Perryville, Missouri, died January 5, 1992 at age 78. Dr. Feltz was honored recently at the Missouri State Medical Association's annual convention. In his 50-year medical career, he delivered more than 3,000 babies throughout Perry County in a private practice which included day and night house calls. A native of Perryville, Dr. Feltz graduated from St. Vincent's High School and attended St. Louis University, receiving his M.D. degree in 1941. Following service with the Army Medical Corps in the Pacific Islands and Australia, Dr. Feltz returned to Perryville in 1946 to practice medicine. He was a member of St. Louis University Alumni Association, American Medical Association, and Southern Medical Association.

■ **John L. Washburn, M.D.**, Versailles, Missouri, died January 5, 1992 at age 81. Dr. Washburn received his medical degree from Northwestern University, Chicago in 1934. He practiced medicine in Versailles, Missouri from 1935 until semi-retirement in 1977, finally retiring in 1981. He helped establish the Morgan County Health Center, serving on its board of directors for 15 years. He was a member of Lake of the Ozarks Medical Society, Missouri State Medical Association and the American Medical Association.

■ **Wayne A. Simril, M.D.**, died January 2, 1992 at age 70. Dr. Simril was the head of radiology at Shriners Hospital for Crippled Children for 37 years. He was also on staff at Lutheran, Barnes and Faith hospitals and at the Mallinckrodt Institute of Radiology. He received his medical degree from Washington University School of Medicine, and was an instructor in the anatomy department there while serving his residency at the Mallinckrodt Institute of Radiology. He served as a captain in the Army Medical Corps during World War II, and in 1952, he and Dr. Wendell Scott established the Scott Radiological Group. Author of numerous papers on hip and bone diseases in children, Dr. Simril was one of the first allied specialists to be elected a member of the American Academy of Orthopedic Surgeons. He was a member of the American Medical Association, the Southern Medical Society, and the St. Louis Metropolitan Medical Society.

■ **Axel R. Gronau, M.D.**, died November 27, 1991 at age 81. Dr. Gronau was a native of Marienburg, Germany. He attended the University of Gottingen in Germany and received his medical degree from the University of Naples in Italy. He came to St. Louis in 1936, joining the department of bacteriology at the Washington University School of Medicine, and later joined the staff at Koch Hospital. After serving in the U.S. Army during World War II, Dr. Gronau practiced privately before returning to Koch Hospital during the 1950's, retiring from Koch in 1978 when he was made clinical instructor emeritus at Washington University. He was a member of St. Louis Metropolitan Medical Society.

CORRECTION:

In the January 1992 issue, the late Dr. Herbert M. Arnold's graduation year from medical school was listed incorrectly. The correct year was 1947. *Missouri Medicine* regrets this error.

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STUDENT LOAN DEFERMENT ELIMINATED, BUT FIGHT CONTINUES

The House and Senate delivered the final blow to student loan deferments as the 1991 Congressional sessions came to a close.

In separate action on the Reauthorization of the 1965 Higher Education Act, the House Education and Labor Committee eliminated current student loan deferments for resident physicians by approving H.R. 3553, while the Senate Labor Committee approved S.1150, which would eliminate loan deferments as of 1993. The Senate bill also restricts the availability of forbearance to residents whose debt exceeds 20% of their income and limits the maximum forbearance period to three years (from 10). In addition to medical residents, the action will affect groups such as the armed services, Peace Corps, and VISTA.

The AMA is involved in a coalition of specialty, medical school, and other educational groups in a battle to regain the student loan deferment and forbearance provisions.

In an urgent call-to-action, the AMA is asking medical students, resident physicians, and their spouses to encourage Congress to continue loan deferments for resident physicians; and to restore forbearance in Senate bill to the full 10-year period regardless of income. Congressmen can be reached by calling 202/224-3121, and leaving a message with the legislative aide.

STUDENT LOAN UPDATE - - - KEEP THOSE CARDS AND LETTERS COMING!

Legislators still find it extremely difficult to grasp the fact that medical residents are not "rich doctors" and that most residents have exorbitant student loan debts that must be paid NOW, not when their salaries are more commensurate with their debt load.

Take time TODAY to write your elected officials a personal note. Outline your actual financial situation by including the following:

Your current debt level;

Your anticipated debt level at the completion of your residency training;

Your estimated monthly repayment amounts, providing a relative comparison by including a percentage against your "take home" pay;

Any personal vignettes about your present financial picture;

Vignettes about how your level of debt has affected your specialty choice, your residency choice, or your geographical choice for a future practice (Since shortages of primary care, rural physicians in Missouri are considered at "Crisis Proportions", this is a key piece of information to include).

In addition, be sure to include your request a return to or an increase in current deferment/forbearance periods. Finally, take time to thank your elected officials for their time.

Senators:
The Honorable Christopher Bond / John C. Danforth
C/O
United States Senate
Washington, DC 20510

Representatives:

The Honorable
Bill Clay - C-1
Joan Kelly Horn - C-2
Dick Gephardt - C-3
Ike Skelton - C-4
Alan Wheat - C-5
Tom Coleman - C-6
Mel Hancock - C-7
Bill Emerson - C-8
Harold Volkmer - C-9
C/O
United States House of Representatives
Washington, DC 20515

Joining a Partnership or Group Practice Seminar

This seminar will be held at the MSMA annual convention and is sponsored by the MSMA-RPS. Its cost is $75 for non-members of the MSMA and $35 for MSMA members. This is a significant reduction in price from the regular cost for attending this seminar.

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■ **Lillian C. Alderman, M.D.**, 5400 N. Oak Trafficway, Suite 206, Kansas City, MO 64118, has become a member of the Clay-Platte County Medical Society. Dr. Alderman received her baccalaureate degree from the University of Colorado, Boulder and obtained her medical degree from the University of Kansas School of Medicine, Lawrence-Kansas City in 1984. She completed her residency at Kansas University Medical Center. Dr. Alderman specializes in obstetrics and gynecology.

■ **Stephen M. Benz, M.D.**, 12805 Canterbury Farms Dr., St. Louis, MO 63128, has become a member of the Missouri State Medical Association. Dr. Benz received his baccalaureate degree from Washington University, St. Louis and obtained his medical degree from St. Louis University School of Medicine, St. Louis in 1982. He completed his residency at the University of Kentucky Medical Center, Lexington. Dr. Benz specializes in orthopaedic surgery.

■ **Cynthia L. Brown, M.D.**, 2875 James Blvd, Poplar Bluff, MO 63901, has become a member of the Butler-Wayne-Ripley-Carter County Medical Society. Dr. Brown received her baccalaureate degree from Fordham University, New York, NY and obtained her medical degree from Mount Sinai School of Medicine, New York, NY in 1982. She completed her residency at State University of New York at Stony Brook, University Hospital. Dr. Brown specializes in obstetrics and gynecology.

■ **Fred M. Caldwell, M.D.**, 1901 Sunset, Poplar Bluff, MO 63901, has become a member of the Butler-Wayne-Ripley-Carter County Medical Society. Dr. Caldwell received his baccalaureate degree from University of Missouri, Kansas City and obtained his medical degree from University of Missouri School of Medicine, Kansas City in 1984. He completed his residency at University of Arkansas School of Medicine, Little Rock. Dr. Caldwell specializes in internal medicine/pediatrics.

■ **Paul R. Cook, M.D.**, MA314, 1 Hospital Drive, Columbia, MO 65212, has become a member of the Boone County Medical Society. Dr. Cook received his baccalaureate degree from the University of Oklahoma, Norman and obtained his medical degree from the University of Oklahoma College of Medicine, Oklahoma City in 1980. He completed his residency at Truman Memorial VA Hospital, Columbia, MO. Dr. Cook specializes in otolaryngology.

■ **Manuel S. Danganan, M.D.**, 1208 Walnut St., Doniphan, MO 63935, has become a member of the Butler-Wayne-Ripley-Carter County Medical Society. Dr. Danganan received his baccalaureate degree from University of Santo Thomas, Manila, Philippines and obtained his medical degree from Faculty of Medicine and Surgery, University of Santo Thomas, Manila in 1957. He completed his residency at Saint Agnes Hospital, Raleigh, NC. Dr. Danganan specializes in general practice.

■ **Larry F. Frevert, M.D.**, 6650 Troost, Suite 103, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Frevert received his baccalaureate degree from Texas Christian University and obtained his medical degree from University of Missouri School of Medicine, Columbia in 1985. He completed his residency at the University of Arkansas. Dr. Frevert specializes in orthopaedics.

■ **John M. Goldberg, M.D.**, 6724 Troost, Suite 710, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Goldberg received his baccalaureate degree from Tulane University, New Orleans and obtained his medical degree from the University of Kansas School of Medicine, Lawrence-Kansas City in 1987. He completed his residency at the University of Kansas Medical Center. Dr. Goldberg specializes in internal medicine.

■ **Robert R. Hausam, M.D.**, P.O. Box 1254, Sedalia, MO 65301, has become a member of the Pettis County Medical Society. Dr. Hausam received his baccalaureate degree from the University of Missouri and obtained his medical degree from the University of Missouri School of Medicine, Columbia in 1984. He completed his residency at the University of Utah. Dr. Hausam specializes in family practice.

■ **Andrew R. Scott, M.D.**, 6675 Holmes, Suite 400, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Scott received his baccalaureate degree from the University of Oklahoma and obtained his medical degree from the University of Kansas School of Medicine, Lawrence-Kansas City in 1983. He completed his residency at Duke University Medical Center, Durham, NC. Dr. Scott specializes in orthopaedic surgery/sports medicine.

Continued on page 182

NEW MEMBERS

Continued from page 181

■ **John M. Yates, M.D.**, 200 St. Mary's Medical Plaza, Suite 204, Jefferson City, MO 65101, has become a member of the Cole County Medical Society. Dr. Yates received his baccalaureate degree from Oklahoma State University and obtained his medical degree from the University of Oklahoma College of Medicine, Oklahoma City in 1973. He completed his residency at Letterman Army Medical Center, San Francisco. Dr. Yates specializes in psychiatry.

RESIDENTS

Eduardo D. Flores, M.D. *cardiology*
Randall K. Gibb, M.D. *obstetrics and gynecology*
Kurt A. Henry, M.D. *internal medicine*
Bryon L. Limmer, M.D. *dermatology*
Tammy K. Neblock, M.D. *general surgery*
Steven J. Prstojevich, M.D. *oral/maxillofacial surgery*
Walter Rayford, M.D. *general surgery*
Robert E. Schroeder, M.D. *pediatrics*
Juliette A. Stell, M.D. *obstetrics and gynecology*

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nizatidine
150 mg b.i.d.

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[1] Data on file, Lilly Research Laboratories.

See accompanying page for prescribing information.

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NZ-2947-B-249304

AXID®

nizatidine capsules

Brief Summary. Consult the package insert for complete prescribing information.

Indications and Usage: 1. *Active duodenal ulcer*—for up to 8 weeks of treatment at a dosage of 300 mg h.s. or 150 mg b.i.d. Most patients heal within 4 weeks.

2. *Maintenance therapy*—for healed duodenal ulcer patients at a dosage of 150 mg h.s. at bedtime. The consequences of therapy with Axid for longer than 1 year are not known.

3. *Gastroesophageal reflux disease (GERD)*—for up to 12 weeks of treatment of endoscopically diagnosed esophagitis, including erosive and ulcerative esophagitis, and associated heartburn at a dosage of 150 mg b.i.d.

Contraindication: Known hypersensitivity to the drug. Because cross sensitivity in this class of compounds has been observed, H_2-receptor antagonists, including Axid, should not be administered to patients with a history of hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests—False-positive tests for urobilinogen with Multistix® may occur during therapy.

Drug Interactions—No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups.The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of anti-androgenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated, however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

PV 2093 AMP [101591]

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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221, November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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St. Louis University School of Medicine is now testing its newest AIDS vaccine in women to evaluate how many shots of a particular vaccine must be given to rapidly stimulate an immune response. This preliminary study is being done in low risk, uninfected and nonpregnant women. It is the fifth human AIDS vaccine trial authorized by the FDA at SLU, which is one of two institutions in the nation testing this particular protocol.

Sophia M. Chung, M.D., a neuro-ophthalmologist in the department of ophthalmology, has linked smoking with the gradual loss of vision associated with aging. Anterior ischemic optic neuropathy (AION), a disease that occurs when the blood supply to the optic nerve is reduced, results in dimming, blurring and sudden vision loss. In a study of 148 patients over a 10-year period, Dr. Chung discovered that the onset of the disease was seven years earlier in smokers than in nonsmokers and the risk for developing AION dramatically decreases when a person stops smoking.

The Souers Stroke Institute at St. Louis University School of Medicine received a Master Agreement from the National Institute of Neurological Disorders and Stroke of the National Institutes of Health. The three-year agreement prequalifies the institute to participate in specialized cerebrovascular clinical research projects sponsored by NIH.

Duane P. Grandgenett, Ph.D., professor of molecular virology, received a five-year grant from the National Institute of Allergy and Infectious Diseases. Dr. Grandgenett will use the award to continue research into the reproductive process of retroviruses.

Carmine J. Coscia, Ph.D., professor of biochemistry and molecular biology received the James A. Shannon Directors Award from the National Health Institutes of Health. Dr. Coscia is studying the regulation of plant alkaloids.

David J. Schanzlin, M.D., chairman of the department of ophthalmology and CEO of Bethesda Eye Institute received a grant from Research to Prevent Blindness.

Thomas Aceto Jr., M.D., professor of pediatrics in the division of endocrinology received a grant from Genentech Inc. to further his research of the use of biosynthetic human growth hormone.

Richard D. Bucholz, M.D., associate professor of surgery in the division of neurosurgery received a two-year grant from Pfizer/Infusaid Corporation of Norwood, Mass. Dr. Bucholz is researching the antispasmodic drug, Baclofen.

Washington University

Researchers at Washington School of Medicine may have found why people with abnormal forms of a cholesterol-carrying protein have low cholesterol levels. A study found that people with genetic mutations of apolipoprotein-B (Apo-B) missing one particular segment may cause liver cells to remove cholesterol from the blood more efficiently, leaving blood cholesterol levels low. These mutations may also cause newly synthesized cholesterol to be released into the bloodstream more slowly than normal. The finding may eventually help explain why some people are genetically prone to high cholesterol levels.

Milton J. Schlesinger, M.D., a professor of molecular microbiology, directed research which developed a compound 95 percent effective at blocking the spread of a laboratory strain of influenza virus. The compound, consisting of a small chain of amino acids, disables the spikes the virus uses to attach to healthy cells. Without the spikes, the virus is immobilized and will be killed by the immune system. Dr. Schlesinger is currently studying the use of this approach on other viruses, including rabies, herpes and HIV.

UNIVERSITY OF MISSOURI-COLUMBIA

MU is combining Department of Engineering research efforts with medicine.

Han Bao, Ph.D., professor of industrial engineering, using a grant sponsored by the Department of Veteran's Affairs and NASA, has developed a prototype machine called "Shoefitter." This machine will significantly decrease the $300 to $500 price people with irregular or injured feet pay for custom-made shoes by cutting 75 percent of time needed to produce the final cast of the foot around which the shoe is made. It takes two days to make casts using manual methods. Shoefitter only takes four hours for two feet: one hour to scan the feet with laser and process the data, another hour to design the shoe patterns on a personal computer and two more hours to machine the final casts, including setup time to prepare the machinery. Dr. Bao says the process will be made even faster with better equipment.

Hirotsugu Yasuda, Ph.D., professor of chemical engineering and director of the Center for Surface Science Plasma Technology is researching improvements for bone cement. To make cement visible to X-ray, a special pigment is added that detracts from the strength of the cement. By exposing the pigment to an ionized gas plasma of hydrogen, oxygen and methane he has improved the interface between the cement and bone. Dr. Yasuda says preliminary tests found the treated pigment strength of the cement to improve to one hundred percent or more of the cement without pigment.

Joint research efforts by **William Carson, Ph.D.,and Roger Duffield, Ph.D.,** professors of mechanical engineering and **Robert Gaines, M.D.,** an orthopedic surgeon at the University Hospital and Clinics, have contributed to successful treatment of spinal fractures. In laboratory tests the team analyzed patterns of stress transfer through spinal implants and thoroughly investigated the fundamentals of spinal fracture repair. The testing has enabled surgeons to select the best components, reducing the likelihood of failure.

Dr. Shelden

Russell D. Shelden, M.D., and his wife, Mary, are the benefactors of the Russell D. Shelden, M.D., Distinguished Professorship of Anesthesiology. A faculty recipient from the department will be selected to hold the professorship once the endowment is completed. Shelden has been active in local, state and national medical orgaizations and is a past president of the Missouri Society of Anesthesiologists and the Kansas City Society of Anesthesiologists.

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MARCH

3 - 4 — ***St. Louis*** **Transesophageal Echocardiography Course** - Location: Saint Louis University Medical Center. Contact: CME Office, SLU, 1402 S. Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6— ***St. Louis*** **Host-Pathogen Interactions in Microbial Pathogensis** - Location: 3rd floor Auditorium, Children's Hospital. Contact: Department of Pediatrics, Washington University School of Medicine.

6 — ***St. Louis*** **Controversies in the Treatment of Early Breast Cancer and in the Treatment of Gynecological Cancer** - Location: The Ritz-Carlton Hotel, St. Louis, MO. Contact: St. Louis University Medical Center CME Office at 1-800-553-2712 or 314-577-8167.

6 — ***St. Louis*** **Current Perspectives in Cancer Therapy, The Multi-modal Approach** - Location: The Ritz-Carlton Hotel, St. Louis, MO. Contact: CME Office, SLU , 1402 South Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 - 7 — ***St. Louis*** **Advanced Trauma Life Support Provider** - Location: Margaret McCormick Doisy Learning Resources Center. Contact: CME Office, SLU, 1402 South Grand Blvd., St. Louis, MO 63104. 800/553-2712.

6 - 8 — ***Columbia*** **Family Practice Update** - Location: UMC School of Medicine, Columbia, MO. Contact: Barry Kling, CME Office, MA215 Health Sciences Center. 314/882-4105.

7 — ***St. Louis*** **Flexible Sigmoidoscopy** - Location: DePaul Health Center, St. Louis Seton Auditorium and Classroom 3. Contact: Sheri Leppanen, Depaul Health Center, (314) 344-6000, ext. 2339.

9 — ***St. Louis*** **CT of the Lung Parenchyma** - Location: Mallinckrodt Institute of Radiology. Contact: R. Gilbert Jost, M.D., Mallinckrodt Institute of Radiology, 510 S. Kingshighway Boulevard, St. Louis, MO 63110. 314/362-7130.

13 — ***St. Louis*** **Orbit and Eyelid Surgery** - Location: Margaret McCormick Doisy Learning Resources Center, Saint Louis University. Contact: CME Office, SLU, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

15 - 20 — ***Park City, UT*** **Difficult Situations in Clinical Practice (14th Annual Winter Psychiatry Conference)**- Location: Park City, Utah. Contact: The Menninger Clinic, Box 829, Topeka, KS 66601-0829.

27 — ***St. Louis*** **Tenth Annual Geriatric Symposium** - Location: Breckenridge Frontenac, Contact: CME Office, St. Louis University Medical Center, 1412 South Grand Blvd., St. Louis, MO 63104. 314/577-8167.

APRIL

4 — *St. Louis*: **Liver Transplantation for the Primary Care Physician**. Category 1 credit. Contact Office of CME, St. Louis University Medical Center, 314/577-8167 or 800/553-2712.

7 to 9 — *St. Louis*: **Introduction to Doppler Echocardiography**. Category 1 credit. Contact Diane Reid, St. Louis University Medical Center, 314/577-8897.

13 to 17 — *St. Louis:* **Neuro CT Program**. Category 1 credit. Contact Louis A. Gilula, M.D., Visiting Fellowship Program, Mallinckrodt Institute of Radiology, 510 S. Kingshighway Blvd., St. Louis, MO 63110-1076. PH: 314/362-2916.

20 to 24 — *St. Louis*: **Medicolegal Death Investigator Training Course**. Category 1 credit. Contact Mary Fran Ernst, St. Louis University Medical Center, 314/522-640 ext. 6509.

20 to 24 — *St. Louis*: **Body CT Program**. Category 1 credit. Contact Louis A. Gilula, M.D., Visiting Fellowship Program, Mallinckrodt Institute of Radiology, 510 S. Kingshighway Blvd., St. Louis, MO 63110-1076. PH: 314/362-2916.

24 to 26 — *St. Louis*: **Practical Anatomy Surgical Technique Workshop, Spinal Fracture Fixation**. Category 1 credit. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

25 — *St. Louis*: **3rd Annual Course: Management of Colon and Rectal Disease in 1992**. Category 1 credit. Contact Office of CME, St. Louis University Medical Center, 314/577-8167 or 800/553-2712.

27 to May 1 — *St. Louis*: **Magnetic Resonance Imaging of Head and Body**. Category 1 credit. Contact Louis A. Gilula, M.D., Visiting Fellowship Program, Mallinckrodt Institute of Radiology, 510 S. Kingshighway Blvd., St. Louis, MO 63110-1076. PH: 314/362-2916.

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References should be cited in numerical order in the text and arranged at the end of the article in the same order in which they are cited. The reference should give name of author, title of article, name of periodical, volume number, initial page number and year. Authors are responsible for reference accuracy.

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Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

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Owned and edited by the Missouri State Medical Association. *Missouri Medicine*, Journal of the Missouri State Medical Association, is published monthly at 113 Madison, Jefferson City, Missouri 65102. Second-Class postage paid at Jefferson City, Missouri and at additional mailing offices. Postmaster: Send address changes to Missouri State Medical Association, c/o Membership Dept. P.O. Box 1028, Jefferson City, MO 65102.

The subscription price per year for non-members is $20.00. The subscription price for members is included in the annual membership dues. The international subscription price is $40.00. Single copies are $4.00 and the cost of the annual August Directory issue is $15.00.

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Printed by the Ovid Bell Press, Inc., Fulton, Missouri 65251.

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MONETAMED PROFILE

THOMAS P. DOYLE, M.D.

HOME: Springfield, Missouri.

AGE: 36.

SPECIALTY: Orthopedic Surgery.

MEDICAL COLLEGE: University of Missouri, 1978

PRACTICE: "I recently purchased an established but declining sole practitioner office from a retiring physician. It's a risk, but they say that great deeds are usually wrought at great risk."

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MISSOURI MEDICINE
Journal of the Missouri State Medical Association

Vol. 89, No. 4 April 1992

April's Cover:

We are pleased to feature an oil portrait titled "Meditation" by Donald K. Piper, M.D.. Dr. Piper practiced medicine in the Kansas City area for 35 years before retiring. George R. Wempe, M.D., the subject, practiced in Nodaway County for nearly 40 years. Their friendship has stretched over 70 years.

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"...More on this story, and tape at 10."

By J. Regan Thomas, M.D., Editor

Many of the perceptions the public forms about physicians and medicine are a direct result of what they see on television. The television news media are the dominant source of information for much of the American public today. The physician community finds itself more frequently interacting with representatives of that medium and often feels ill at ease doing so. Whether being interviewed as part of a news story, or participating in a question and answer session format, there are techniques and suggestions which will enhance that experience.

It is difficult to make many of us understand that most medically related news stories are not controversial or an attempt to do undercover investigative reporting. Instead they are typically an attempt to be informative and your interview is simply a credible way to confirm or shed light on a specific story. So, rule one is to relax and don't assume the reporter is "out to get you." That attitude may make you appear defensive or negative and will not afford the optimum setting for your message.

The initial process for developing a successful interview actually begins before you meet the reporter. Anticipate likely questions on the topic. Practice your answers in advance to feel more comfortable and confident. Be prepared to keep your answers brief and to the point. Remember, television reporting is a time-limited medium and brief "sound bites" are what make it on the air. The ability to be strategic with your comments, both in content and length enables you to better control the information eventually being aired.

Your appearance and clothing during an interview will also affect the way your message is perceived. The basic rule is to dress conservatively. Newer cameras and technology make some of the old rules less important, i.e. blue shirts instead of white. However, men should tend to wear dark suits with a light, solid color shirt. Women may prefer a suit, but if a dress is worn it should be a solid or subtle print. Avoid stripes, bright plaids, or bold prints. Don't wear flashy jewelry, dangling earrings and bracelets, or large lapel pins. Never wear light sensitive glasses that darken under the lights. It has been said that "what a speaker wears should never overpower what he or she has to say." Television tends to magnify these aspects even more so take a tip from the professionals and dress in a conservative "news anchor" fashion.

The next stage begins when you are actually with the reporter or interviewer. Try to engage the reporter in conversation prior to the interview. This may give insight to the direction of the interview as well as establish some rapport.

As the interview begins, ignore the camera and keep good eye contact with the interviewer. At the same time, although the reporter is asking the question, remember as you answer you are talking to the general public. Answer in succinct concise statements and wait silently for the next question. Listen carefully to the questions. If you "flub" a line or mistake your answer, don't hesitate to restate your comment.

Be aware of your body language. Do your best to avoid gestures and expressions that are interpreted as negative or convey tension or nervousness. Common examples of gestures to avoid include toying with pencils or glasses, "drumming" the table top, gripping the sides of the chairs or tables, and tightening and loosening of the jaw. Closing the eyes when speaking or shifting the eyes upward may convey impatience or discomfort. Absolutely avoid rocking or swaying in a swivel chair during the session.

Most consultants would agree that you should scrupulously avoid acronyms, or technical jargon. "Plain English" is the goal during the interview. Avoid beginning with trite or cliche phrases like "I'm glad you asked that," or "in the medical field we like to say ... " Likewise, avoid speech mannerisms such as "uh", "er-ah" or "you know."

Continued on page 204

Editor's Desk

Continued from page 203

The greatest fear for most physicians being interviewed is to encounter the hostile or antagonistic interviewer. There are several suggestions to be followed if this situation is encountered. First, feel free to correct a misstatement by the interviewer and don't let them put words in your mouth. Disagree tactfully or correct the facts and go on with your statement. If the question is posed as a choice of answers, both of which are wrong, simply set the record straight with your opinion making the information as positive as possible. Feel free to discount false information.

Always remember, you are in someone else's territory and you will never win a confrontation with the media. Who does the editing and selects the quotes to be aired? The best you can do is be factual, truthful, and honest. Always stay neutral in attitude, courteous in your manner, and pleasant in demeanor. That doesn't mean you can not be controversial or express disagreement. However, if you seem to be attacking the media or challenging the interviewer, you will invariably lose the confrontation. Just try to think who "wins" most of the confrontations, Ted Koppel or the other guys on the interview screen? Never let anything get you hostile while being interviewed.

Two final pearls of advice. First, never hesitate to say "I don't know" and offer to get back to them with the information. If you give misinformation, it is on tape **forever**. Second, "off the record" comments and conversations with reporters do not exist. If you don't want it on the air - don't say it.

Dealing with the media is increasingly inevitable. Like all skills, this is one that can be developed with practice and some common sense. Like it or not, it is the most influential source of information in our society. Television's tremendous impact can be used to dispense relevant, accurate, medical information, but only if medicine learns to skillfully use the medium. ■

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Dear Editor:

I am once again reminded of the penetrating analysis of the PRO by Dr. John K. Layle in the last issue of *Missouri Medicine*. Today, a few weeks later, I received "PROMO", characterized as the newsletter of the Missouri Patient Care Review Foundation. This was the January 1992 issue.

On page two under the heading "Medicine Admission Policies Clarified" the last paragraph is very instructive. I quote, verbatim, "From time to time, providers will request admission determinations via the telephone or during regional meetings. Although we encourage continuing dialogue, case-specific determinations cannot be made on a concurrent basis, with time frames for responses and final determinations enforced as outlined in the PRO manual." The prior paragraph indicates that only retrospective review is accomplished by the PRO.

This clearly explains that this is only an after-the-fact review by an organization afraid to go ahead and make the hard decisions "up front." No person or organization who acts this way can have the respect of practicing physicians.

Although Dr. Layle has established beyond any reasonable doubt that the PRO is a retrospective quack, even if it were not a quack but a capable peer review, PRO remains only a retrospective nay-sayer. Society in general and our politicians in particular do not deserve the cooperation on physicians or organized medicine for the PRO.

C. Keith Whittaker, Kansas City

Dear Editor:

Health insurance companies and their managers or administrators in our area have evolved the practice of calling the attending physician allegedly to get more information about why the hospitalized patient stays in the hospital, but practically put pressure on the patient and physician for early discharge from the hospital. I have felt that these conversations compromise my primary function as a patient advocate.

Recently I have adopted and implemented the policy of refusing to speak with an insurance company or its representative unless the patient or a patient-surrogate (a family member, for example) is simultaneously on the line, on a conference call. We have the capability of making conference calls in our office. This has served the purpose of incorporating the patient into all health decisions, something which is valuable, I feel; has made the insurance company deal with their own insureds; and has been astonishingly enlightening to me and to the patient. Insurance companies either refuse to talk under these circumstances (to their own insured!) or adopt a hard line and are insistent for "information." I suggest that all physicians should adopt this policy. Insurance companies should not be afraid of talking to their own insureds. Physicians likewise should not be caught in the middle in an ethical no-win situation.

I intend to continue this policy and practice and urge my colleagues to consider it also.

C. Keith Whittaker, M.D., Kansas City

Dear Editor:

I would like to respond to the Boyarsky plea for a new specialty, Thanatology. Certainly the Danforth-Moynihan Act has helped physicians and hospitals confront the problems of the dying patients as well as the need to consider end of life options. The hope is that full discussions will take place to explore and elucidate the feeling and desires of our patients. At the very least, all of us will be better educated to face these very difficult issues.

However, the need for yet another medical specialty seems far from clear. Every hospital certainly should have an ethics committee; and those I am familiar with have a membership identical with the consultants to be "shepherded" by the new specialist.

I would feel strongly that the central figure in this scenario should be the patient's own physician, family practitioner, internist, etc. In that setting, empathy and intimacy would be surely combined with a detailed understanding of the family situation. The ethics committee would serve in the role of consultant both prior to hospitalization and for any in hospital problems. Such committees usually include the cast of characters listed in the paper.

Therefore, while I applaud the paper's description of the many serious situations that arise so often when "life is ebbing", I would be loath to support the appearance of another super-specialist on the scene. The hope would be that our profession will face these challenges with the devotion, understanding and skill of which it is capable. Each physician then would maintain his or her rightful place as the patient's best advocate.

Richard G. Sisson, M.D., St. Louis

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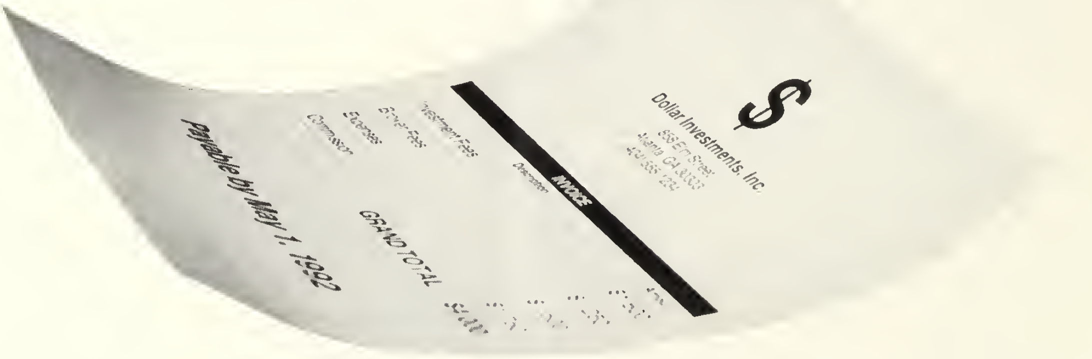
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Hypotension: Symptomatic hypotension was rarely seen in uncomplicated hypertensive patients treated with ACCUPRIL but, as with other ACE inhibitors, it is a possible consequence of therapy in salt/volume depleted patients, such as those previously treated with diuretics or dietary salt restriction or who are on dialysis (see PRECAUTIONS, DRUG INTERACTIONS, and ADVERSE REACTIONS). In controlled studies, syncope was observed in 0.4% of patients (N = 3203); this incidence was similar to that observed for captopril (1%) and enalapril (0.8%).

In patients with concomitant congestive heart failure, with or without associated renal insufficiency, ACE inhibitor therapy may cause excessive hypotension, which may be associated with oliguria or azotemia and, rarely, with acute renal failure and death. In such patients, ACCUPRIL therapy should be started at the recommended dose under close medical supervision. These patients should be followed closely for the first 2 weeks of treatment and whenever the dosage of antihypertensive medication is increased (see DOSAGE AND ADMINISTRATION).

If symptomatic hypotension occurs, the patient should be placed in the supine position and, if necessary, normal saline may be administered intravenously. A transient hypotensive response is not a contraindication to further doses; however, lower doses of ACCUPRIL or reduced concomitant diuretic therapy should be considered.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression rarely in patients with uncomplicated hypertension, but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease such as systemic lupus erythematosus or scleroderma. Agranulocytosis did occur during ACCUPRIL treatment in one patient with a history of neutropenia during previous captopril therapy. Available data from clinical trials of ACCUPRIL are insufficient to show that, in patients without prior reactions to other ACE inhibitors, ACCUPRIL does not cause agranulocytosis at similar rates. As with other ACE inhibitors, periodic monitoring of white blood cell counts in patients with collagen vascular disease and/or renal disease should be considered.

Fetal/Neonatal morbidity and mortality: ACE inhibitors, including ACCUPRIL, can cause fetal and neonatal morbidity and mortality when administered to pregnant women.

When ACE inhibitors have been used during the second and third trimesters of pregnancy, there have been reports of hypotension, renal failure, skull hypoplasia, and death. Oligohydramnios has also been reported, presumably resulting from decreased fetal renal function; oligohydramnios has been associated with fetal limb contractures, craniofacial deformities, hypoplastic lung development, and intrauterine growth retardation.

Prematurity and patent ductus arteriosus have been reported, although it is not clear whether these occurrences were due to the ACE-inhibitor exposure or to the mother's underlying disease. It is not known whether exposure limited to the first trimester can adversely affect fetal outcome.

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A patient who becomes pregnant while taking ACE inhibitors, or who takes ACE inhibitors when already pregnant, should be apprised of the potential hazard to her fetus. If she continues to receive ACE inhibitors during the second or third trimester of pregnancy, frequent ultrasound examinations should be performed to look for oligohydramnios. When oligohydramnios is found, ACE inhibitors should generally be discontinued.

Infants with histories of in utero exposure to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion. Hemodialysis and peritoneal dialysis have little effect on the elimination of quinapril and quinaprilat.

No fetotoxic or teratogenic effects were observed in rats at quinapril doses as high as 300 mg/kg/day (180 and 30 times the maximum daily human dose when based on mg/kg and mg/m², respectively), despite maternal toxicity at 150 mg/kg/day. Tested later in gestation and during lactation, reduced offspring body weight was seen at ≥25 mg/kg/day, and changes in renal histology (juxtaglomerular cell hypertrophy, tubular/pelvic dilation, glomerulosclerosis) were observed both in dams and offspring treated with 150 mg/kg/day. Quinapril was not teratogenic in the rabbit; however, as noted with other ACE inhibitors, maternal toxicity and embryotoxicity were seen in some rabbits at quinapril doses as low as 0.5 mg/kg/day (one time the recommended human dose) and 1.0 mg/kg/day, respectively.

PRECAUTIONS

General

Impaired renal function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including ACCUPRIL, may be associated with oliguria and/or progressive azotemia and rarely acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine have been observed in some patients following ACE inhibitor therapy. These increases were almost always reversible upon discontinuation of the ACE inhibitor and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some hypertensive patients with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when ACCUPRIL has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of any diuretic and/or ACCUPRIL may be required.

Evaluation of hypertensive patients should always include assessment of renal function (see DOSAGE AND ADMINISTRATION).

Hyperkalemia and potassium-sparing diuretics: In clinical trials, hyperkalemia (serum potassium ≥5.8 mmol/L) occurred in approximately 2% of patients receiving ACCUPRIL. In most cases, elevated serum potassium levels were isolated values which resolved despite continued therapy. Less than 0.1% of patients discontinued therapy due to hyperkalemia. Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with ACCUPRIL (see PRECAUTIONS, Drug Interactions).

Surgery/anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, ACCUPRIL will block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients

Angioedema: Angioedema, including laryngeal edema, can occur with treatment with ACE inhibitors, especially following the first dose. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to stop taking the drug until they have consulted with their physician (see WARNINGS).

Symptomatic hypotension: Patients should be cautioned that lightheadedness can occur, especially during the first few days of ACCUPRIL therapy, and that it should be reported to a physician. If actual syncope occurs, patients should be told to not take the drug until they have consulted with their physician (see WARNINGS).

All patients should be cautioned that inadequate fluid intake or excessive perspiration, diarrhea, or vomiting can lead to an excessive fall in blood pressure because of reduction in fluid volume, with the same consequences of lightheadedness and possible syncope.

Patients planning to undergo any surgery and/or anesthesia should be told to inform their physician that they are taking an ACE inhibitor.

Hyperkalemia: Patients should be told not to use potassium supplements or salt substitutes containing potassium without consulting their physician (see PRECAUTIONS).

Neutropenia: Patients should be told to report promptly any indication of infection (eg, sore throat, fever) which could be a sign of neutropenia.

NOTE As with many other drugs, certain advice to patients being treated with ACCUPRIL is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions

Concomitant diuretic therapy: As with other ACE inhibitors, patients on diuretics, especially those on recently instituted diuretic therapy, may occasionally experience an excessive reduction of blood pressure after initiation of therapy with ACCUPRIL. The possibility of hypotensive effects with ACCUPRIL may be minimized by either discontinuing the diuretic or cautiously increasing salt intake prior to initiation of treatment with ACCUPRIL. If it is not possible to discontinue the diuretic, the starting dose of quinapril should be reduced (see DOSAGE AND ADMINISTRATION).

Agents increasing serum potassium: Quinapril can attenuate potassium loss caused by thiazide diuretics and increase serum potassium when used alone. If concomitant therapy of ACCUPRIL with potassium-sparing diuretics (eg, spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes is indicated, they should be used with caution along with appropriate monitoring of serum potassium (see PRECAUTIONS).

Tetracycline and other drugs that interact with magnesium: Simultaneous administration of tetracycline with ACCUPRIL reduced the absorption of tetracycline by approximately 28% to 37%, possibly due to the high magnesium content in ACCUPRIL tablets. This interaction should be considered if coprescribing ACCUPRIL and tetracycline or other drugs that interact with magnesium.

Lithium: Increased serum lithium levels and symptoms of lithium toxicity have been reported in patients receiving concomitant lithium and ACE inhibitor therapy. These drugs should be co-administered with caution, and frequent monitoring of serum lithium levels is recommended. If a diuretic is also used, it may increase the risk of lithium toxicity

Other agents: Drug interaction studies of ACCUPRIL with other agents showed:

- Multiple dose therapy with propranolol or cimetidine has no effect on the pharmacokinetics of single doses of ACCUPRIL.
- The anticoagulant effect of a single dose of warfarin (measured by prothrombin time) was not significantly changed by quinapril coadministration twice-daily.
- ACCUPRIL treatment did not affect the pharmacokinetics of digoxin.
- No pharmacokinetic interaction was observed when single doses of ACCUPRIL and hydrochlorothiazide were administered concomitantly

Carcinogenesis, Mutagenesis, Impairment of Fertility

Quinapril hydrochloride was not carcinogenic in mice or rats when given in doses up to 75 or 100 mg/kg/day (50 to 60 times the maximum human daily dose, respectively, on a mg/kg basis and 3.8 to 10 times the maximum human daily dose when based on a mg/m² basis) for 104 weeks. Female rats given the highest dose level had an increased incidence of mesenteric lymph node hemangiomas and skin/subcutaneous lipomas. Neither quinapril nor quinaprilat were mutagenic in the Ames bacterial assay with or without metabolic activation. Quinapril was also negative in the following genetic toxicology studies: *in vitro* mammalian cell point mutation, sister chromatid exchange in cultured mammalian cells, micronucleus test with mice, *in vitro chromosome* aberration with V79 cultured lung cells, and in an *in vivo* cytogenetic study with rat bone marrow. There were no adverse effects on fertility or reproduction in rats at doses up to 100 mg/kg/day (60 and 10 times the maximum daily human dose when based on mg/kg and mg/m², respectively).

Pregnancy

Pregnancy Category D: See WARNINGS, Fetal/Neonatal morbidity and mortality.

Nursing Mothers

It is not known if quinapril or its metabolites are secreted in human milk. Quinapril is secreted to a limited extent, however, in milk of lactating rats (5% or less of the plasma drug concentration was found in rat milk). Because many drugs are secreted in human milk, caution should be exercised when ACCUPRIL is given to a nursing mother.

Geriatric Use

Elderly patients exhibited increased area under the plasma concentration time curve (AUC) and peak levels for quinaprilat compared to values observed in younger patients; this appeared to relate to decreased renal function rather than to age itself. In controlled and uncontrolled studies of ACCUPRIL where 918 (21%) patients were 65 years and older, no overall differences in effectiveness or safety were observed between older and younger patients. However, greater sensitivity of some older individual patients cannot be ruled out.

Pediatric Use

The safety and effectiveness of ACCUPRIL in children have not been established.

ADVERSE REACTIONS

ACCUPRIL has been evaluated for safety in 4960 subjects and patients. Of these, 3203 patients, including 655 elderly patients, participated in controlled clinical trials. ACCUPRIL has been evaluated for long-term safety in over 1400 patients treated for 1 year or more.

Adverse experiences were usually mild and transient.

Discontinuation of therapy because of adverse events was required in 4.7% of patients treated with ACCUPRIL in placebo-controlled hypertension trials.

Adverse experiences probably or possibly related to therapy or of unknown relationship to therapy occurring in 1% or more of the 1563 patients in placebo-controlled hypertension trials who were treated with ACCUPRIL are shown below.

Adverse Events in Placebo-Controlled Trials

	ACCUPRIL (N = 1563) Incidence (Discontinuance)	Placebo (N = 579) Incidence (Discontinuance)
Headache	5.6 (0.7)	10.9 (0.7)
Dizziness	3.9 (0.8)	2.6 (0.2)
Fatigue	2.6 (0.3)	1.0
Coughing	2.0 (0.5)	0.0
Nausea/Vomiting	1.4 (0.3)	1.9 (0.2)
Abdominal Pain	1.0 (0.2)	0.7

Clinical adverse experiences probably or possibly related, or of uncertain relationship to therapy, occurring in 0.5% to 1.0% (except as noted) of the patients treated with ACCUPRIL (with or without concomitant diuretic) in controlled or uncontrolled trials (N = 4397) and less frequent, clinically significant events seen in clinical trials or post-marketing experience (the rarer events are in italics) include (listed by body system):

General: back pain, malaise

Cardiovascular: palpitation, vasodilation, tachycardia, *heart failure*, *hyperkalemia*, *myocardial infarction*, *cerebrovascular accident*, *hypertensive crisis*, *angina pectoris*, *orthostatic hypotension*, *cardiac rhythm disturbances*

Gastrointestinal: dry mouth or throat, constipation, *gastrointestinal hemorrhage*, *pancreatitis*, *abnormal liver function tests*

Nervous/Psychiatric: somnolence, vertigo, syncope, *nervousness*, *depression*

Integumentary: increased sweating, pruritus, *exfoliative dermatitis*, *photosensitivity reaction*

Urogenital: *acute renal failure*

Other: amblyopia, pharyngitis, sinusitis, bronchitis, *agranulocytosis*, *thrombocytopenia*

Angioedema: angioedema has been reported in patients receiving ACCUPRIL (0.1%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with ACCUPRIL should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Clinical Laboratory Test Findings

Hematology: (See WARNINGS)

Hyperkalemia: (See PRECAUTIONS)

Creatinine and blood urea nitrogen: Increases (>1.25 times the upper limit of normal) in serum creatinine and blood urea nitrogen were observed in 2% and 2%, respectively, of patients treated with ACCUPRIL alone. Increases are more likely to occur in patients receiving concomitant diuretic therapy than in those on ACCUPRIL alone. These increases often remit on continued therapy.

* In some patients, the antihypertensive effect may diminish toward the end of the once-daily dosing interval. In such patients, an increase in dosage or twice-daily administration may be warranted.

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Unite and Win

by

Dorothy D. Reister, M.D.
President, Missouri State Medical Association

We have heard and seen through history the effects of "divide and conquer." It is time for our profession to "Unite and Win!" In the past few months we have had some very important beginnings for our march.

In September of 1991 the first Medical-Legal Seminar was held in Columbia, Mo. The Missouri Bar Association took the lead in planning this with input and coordination with the other participants. The seminar was well-attended and felt to be worthwhile; the audience stayed to the very end, asking questions of the panelists and holding informal discussions after the close of the seminar. This year we will hold our second annual seminar on September 12, 1992 in Columbia and it is our turn to lead, again with the cooperation of participating organizations. When we meet this way there is professional as well as social interchange. It opens the way to cooperative, nonconfrontational dialogue from which each of us can help the others. This cooperation could be best expressed in the legislative arena.

Another mutual effort in which we have participated is meeting and planning with the Missouri Association of Osteopathic Physicians and Surgeons. We have had meetings, conversations and after much pressure from both our organizations we finally met with representatives of the Board of Healing Arts on February 20, 1992. This was an all day non-stop meeting in St. Louis. We realize the mutual problems that MSMA and MAOPS have and we were able to openly discuss some of these with the board. The CME requirements were discussed and several suggestions were made to the board. We realize that the board has been told by the Legislature that they have the authority to establish the minimum number of CME hours to be required for license renewals. The board has set this number at the present level of 25 hours Category 1 per year. We asked the board to consider ways in which AMA and AOA awards for CME be acceptable to the board. We discussed the problems of the retired physician relating to CME. At present if the physician does not fulfill CME requirements, he is no longer a licensed physician. The retired physician is deserving of our thanks, our respect, and our honor. We discussed ways in which these physicians could remain our fellow professionals. These suggestions will be taken back to the board. We also considered the treatment physicians receive from investigators from the board. The board needs documentation of abuses and we can all help see that anecdotal evidence is documented. At the meeting we were told that about 80 percent of the cases were closed without action needed as to rules and regulations so their load of investigations could be reduced. We discussed the renewal forms which apparently are being corrected. Proposed legislation was being discussed, but for these three organizations to submit a bill there needs to be continuation of these combined meetings so that it is truly a joint effort. As this shows, we have begun and will continue to keep these efforts going with our professional cooperation leading to solutions.

Another source of attack upon us is the federal government, especially Medicare and HOFA. There are several issues that I would like for you to consider. Many physicians felt that they had inadequate time and information to give an informed decision about continuing to be a participant physician. This is a 12 month contract on our side. Since their information was not available at the time of decision, AMA is petitioning for a six-month re-consideration. Similar delays have occurred with CLIA-68; we do not have all the regulations; yet we are supposed to be obeying them. We repeatedly see the "double standard" rear its ugly head.

So now it is time to assert our rights. The cooperation now started between our professional organizations MUST continue. Those first steps are the hardest. Our battle cry can now be UNITE AND WIN!

PEARL

A Retired Physician's Reflections about a Former Patient

By
John A. Carrier, M.D.

The Christmas Card came back: "Return to sender. Person deceased." A feeling of sadness swept over me. I had been exchanging Christmas cards with Pearl for well over twenty years. I wondered just what had happened to Pearl -when, where, and how did she die?

I am now retired. She had been a patient of mine for many years when I was in practice. I thought back as to how she had first come to me as a patient. I recalled how I had taken care of her husband, Jack; his three sisters and a brother. Two of the sisters were registered nurses whom I had worked with in hospitals and clinics.

Jack was a likeable outgoing character who sometimes he drank too much. During World War II he held down a supervisory job at the St. Louis Ordnance Plant. Occasionally he would report into work with black eyes, abrasions, and contusions plus outlandish stories as to how he had obtained them.

Jack died under my care in a South St. Louis hospital with a cardiac condition.

Pearl continued to see me as a patient on a regular basis. She was a pleasant person to talk to. She always had interesting accounts regarding her personal life and activities. However, she was a difficult patient to manage in that she never wanted to face the facts of her true medical condition. She was a diabetic, but would never believe the lab reports regarding her high blood sugars. Gradually, her liver profile became abnormal, but she refused to have more definitive studies made.

On one occasion during my contacts with Pearl she won $300,000 at the Illinois lottery. In celebrating this she brought me and the office girls some very nice gifts.

Despite the windfall of money she never changed her simple manner of dressing or lifestyle. She lived in a modest apartment, and I do not recall her ever owning an automobile. Her only indulgence was an annual trip to Los Vegas where she played poker "with the best of them." Upon return she would always say she had been a winner!

Pearl was always employed; Sometimes as a clerk in a title-abstract office and other times as an attendant in a hospital radiology department. There she became acquainted with the staff of radiologists. I thought this would be the ideal opportunity to arrange for some ultrasound and CT studies of her abdomen. She became terribly upset at the suggestion, and would never agree to do so.

When I retired in 1986, my wife and I moved to our country place near St. James, Mo. Pearl's Christmas card continued to come until this past year. On one card she indicated she planned a visit to Cuba, Mo. and at the time would come to visit us. That visit never took place.

I have written the record room of the medical clinic she was attending after my retirement. Perhaps they can give me some information regarding Pearl's final days. I hope she didn't suffer too much, and I also hope her attending physician was tolerant of her peculiarities.

As physicians I wonder if we often become too clinical. As I reflect about Pearl and other patients I have known -how important is it that I know all the pathological medical details of their deaths? No doubt I will best remember Pearl as an interesting and loyal patient; a member of a family that I had known so well, and had made from the practice of general and internal medicine such a meaningful life.

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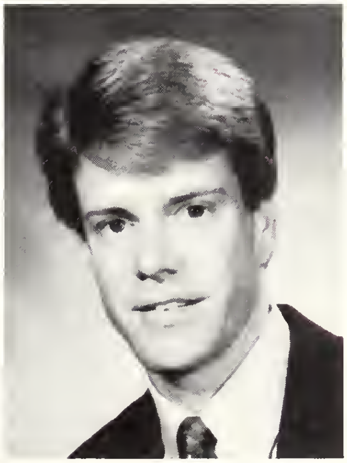
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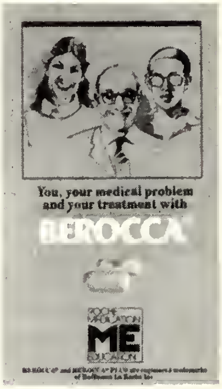
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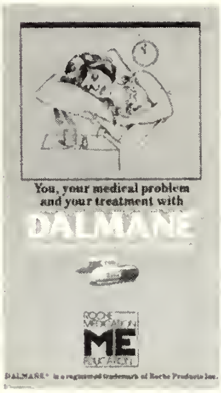
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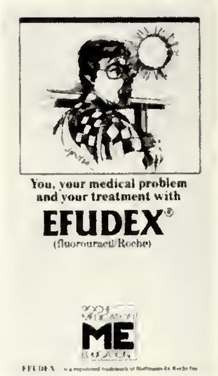
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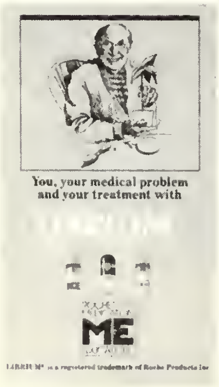
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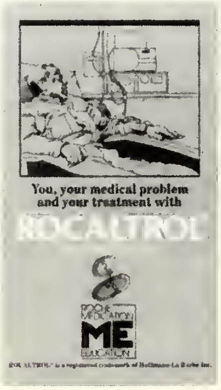
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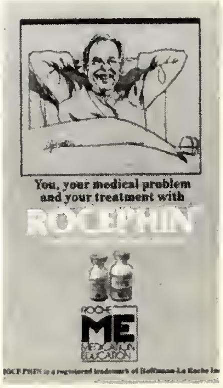
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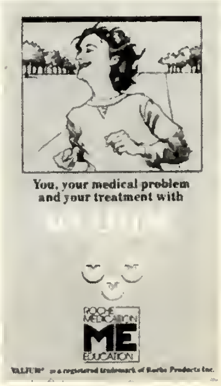
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Dear Dr. Woerth,

I understand that the new hospital codes are different in the 1992 CPT than in previous years. Can you explain the new hospital codes?

Dr. H.M.

Dear Dr. H.M.,

The 1992 CPT includes two types of hospital care codes: initial and subsequent. There is no distinction between new and established patents. The initial hospital care codes are only to be used by the admitting physician. An initial hospital care code includes any medical office visit, nursing home visit, home visit, emergency room visit, or critical care service that result in a hospital admission when the visit/service is provided by the same physician on the same date as the hospital admission. Thus, on the day of a hospital admission it is not appropriate for the physician to also bill for an office visit, nursing home visit, home visit, emergency room visit, or critical care unless that visit/service was unrelated to the hospital admission. For example, if a patient were seen in your office in the morning for removal of a benign lesion and later that same evening the same patient was admitted to the hospital for cardiac arrest, it **would** be appropriate to charge for the office visit and the hospital admission because the hospital admission was unrelated to the patient's problem of benign lesion. It is also appropriate for a physician to charge for an office visit, nursing home visit, home visit, emergency room visit, or critical care even though the patient was admitted to the hospital if **another** physician made the hospital admission later that same day.

Please notice that all three initial hospital care codes included a comprehensive history and physical examination. However, the level of decision making, the severity of the patient's illness, the counseling and/or coordination of care and the time spent with the patient do differ among these three initial hospital care codes. Thus, it is important that the physician document in the patient's medical record those factors that resulted in the rendering of the level 1, 2, or 3 initial hospital care.

When coding for a patient that is placed in an observation area of the hospital and is not admitted to the hospital, use an office visit code and indicate on the claim form, in the box for "place of service", that it is an out-patient service. You will have to use the appropriate level of office visit code for a new or established patient.

There are also three levels of subsequent hospital care. The physician is only required to complete two of the three key components (history, examination and decision making) to use the code. All six of the components of these codes vary but all three codes require the reviewing of medical records, of results of diagnostic studies and of changes in the patient's status. There is a hospital discharge day management code that can be used for the final day of a multiple-day stay. Finally, the subsequent hospital visits are also used for patients in critical care units of the hospital that are not critically ill.

Dear Dr. Woerth,

Frequently we have healthy patients come to our office for counseling with the physician. For example, a patient that is healthy and wants to start an exercise program, diet and/or quit smoking. Can we charge for this counseling time even though the patient is healthy?

Dr. M.M.P.

Dear Dr. M.M.P.,

There are new codes in the 1992 CPT that exactly fit this situation. They are the counseling and/or risk factor reduction intervention codes found on pages 42-43 of the 1992 CPT book. These codes are to be used for healthy individuals and not patients with symptoms or established illnesses. Notice that the codes are broken-down into fifteen minute intervals for individual counseling and thirty minute intervals for groups counseling.

Many of you will be wondering if insurance companies will pay for these risk factor reduction codes. Probably not. Most medical insurance policies do not cover preventive medical services. However, this does not mean that you do not bill for these sevices. If the patient's policy does not cover the service, then generally it is the financial responsibility of the patient to pay the physician directly for the service.

Do you have a question for Dr. Woerth? Include your questions along with your name, address and phone number and send them to:

Janice K. Woerth, Ph.D.
Practice Management
c/o *Missouri Medicine*
P.O. Box 1028
Jefferson City, MO 65102
Or call her direct:
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Although your name will not be used in *Missouri Medicine*, please include your name and phone number so that Dr. Woerth may contact you if she needs additional information to answer your question.

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Change is inevitable

By Mary Hunkeler, MSMAA President

Change is one thing that is inevitable. It goes on in ourselves, our families, and the Auxiliary. Sometimes change becomes hard to see until time passes by for a while. The MSMA Auxiliary has experienced many changes since 1924. And yet our central theme has remained the same, to support medicine and to promote the health and quality of life for all.

Change can be a time of renewal, a time for caring and a time for new thoughts to strengthen old values. It is evidenced by the new leaders who have been asked to lead us in the coming year. And, it will be obvious when auxiliary members come in the 67th Annual Convention, April 9-11, 1992 in St. Louis to recognize the changes and achievements that have taken place in 1991-1992.

At the convention, we will conduct our business in a timely manner to assure continuity and to promote change in our organization. Members will hear reports of officers, county presidents, and committees. We will adopt a budget, elect new officers and convention delegates, and acknowledge outstanding successes among county auxiliaries and special volunteers. New ways to affect change via health projects, legislative issues and membership will be communicated through displays, workshops, and inspirational speakers, including AMA Auxiliary President, Sherry Strebel. Mrs. Strebel will deliver the keynote address and share her commitment to make a difference with a workshop presentation on Family Violence.

Each year, this meeting provides the opportunity for auxiliary members to renew friendships, to share in the fellowship of other physician spouses and to share their common purpose to support medicine and improve the health and quality of health for all.

The time to pass the gavel of leadership to someone else who has new ideas and the commitment to lead our Auxiliary into the future is now. It has been a privilege to serve as president. It is the officers, committee chairmen, county presidents, and the Board of Directors, who were willing to accept change this year. I'm sure they would agree that it's the auxiliary members who really make the difference. I want to extend sincerest thanks to each member for your caring, concern, and commitment to the Auxiliary, to medicine, and the improvement in the quality of health and life for everyone. Thank you.

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DISCIPLINARY ACTIONS

The following is a listing of final disciplinary actions taken by the State Board of Registration for the Healing Arts. The listing includes actions finalized at board meetings held in August and December. This information is made available to the public under the provisions of Section 334.101 RSMo. Names underlined indicate members of the Missouri State Medical Association.

■ **Edward D. Ammala, M.D.**, Fort Leonard Wood, MO.
Board Action: Termination of probation. Effective 8/2/91.

■ **Frank E. Baum, M.D.**, Kansas City, MO. Unprofessional and inappropriate conversations of a sexual nature with a patient. Violation: Section 334.100.2(4) RSMo Supp. 1990
Board Action: License voluntarily surrendered with no reinstatement for seven (7) years. Effective 1/8/92.

■ **William T. Brown, Jr., D.O.**, Independence, MO. Delegated professional responsibilities to persons not qualified to perform them.
Violation: Section 334.100.2(4d)(6)(10)(23) RSMo
Board Action: 180-day suspension -- 150 days stayed, three (3) year probation. Effective 11/12/91.

■ **Patrick T. Conner, M.D.**, Springfield, MO.
Board Action: 1/29/92 - Modification allowing limited prescribing granted to 8/6/90 probationary agreement.

■ **Willie S. Cortez, M.D.**, Lebanon, MO. Dr. Cortez violated the Missouri Board's Amphetamine Rule which instructs the doctor not to prescribe more than a 30-day supply of amphetamine agents per prescription. Additionally, Dr. Cortez' prescribing privileges were restricted by BNDD; his staff privileges were suspended on four occasions for failure to maintain adequate medical records; and he was deficient in required continuing medical education hours.
Violation: Section 334.100.2 RSMo
Board Action: 30-day suspension followed by a five (5) year probation. Effective 1/1/92.

■ **Fernando J. Decastro, M.D.**, St. Louis, MO.
Board Action: 3/1/92 - License reinstated under a seven (7) year probation.

■ **Donald C. Edgren, M.D.**, Cameron, MO.
Board Action: 10/1/91 - Two (2) month suspension followed by a ten (10) year probation for violation of his 7/18/90 probation agreement with the Board by practicing while his license was suspended; not notifying practice settings of his disciplinary status; and his failure to renew his license in a timely fashion.

■ **James Ellegood, M.D.**, Marion, IL. Conviction in U.S. District Court for the Western District of Tennessee of conspiracy to possess with intent to distribute a controlled substance, to wit, 10 kilograms of cocaine and 2400 dosage units of anabolic steroids.
Violation: Section 334.103 RSMo
Board Action: Revocation of Missouri license. Effective 1/3/92.

■ **Paul B. Juergens, M.D.**, Marion, IL.
Board Action: 12/5/91 - Reinstatement denied based on failure to pass required examination.

■ **Mumata Nataraj Kasal, M.D.**, Columbia, MO.
Board Action: 1/27/92 - Temporary license issued on a probationary status wherein she will not apply for renewal or reinstatement of a temporary license to practice in Missouri after the expiration of this agreement.

■ **Walter J. Kutryb, M.D.**, St. Louis, MO.
12/30/91 - Retirement agreement signed in lieu of further investigations or proceedings concerning his license to practice.

■ **Richard Lagueruela, M.D.**, St. Louis, MO. Violation of the drug laws and unprofessional conduct.
Violation: Section 334.100.2(4c)(4h)(5)(13)(14) RSMo. supp. 1988
Board Action: Nine (9) month suspension followed by a three (3) month probation. Effective 8/30/91.

■ **Randall W. Lewis, D.O.**, High Ridge, MO. Personal Impairment.
Board Action: 10/17/91 -- Modification granted to 11/13/89 probationary agreement, new terms to be in effect for seven (7) years.

■ **Barry Moore, M.D.**, Greenville, NC. Revocation of license in North Carolina based on personal, intimate relationships of a sexual nature with patients.
Violation: Section 334.103 RSMo
Board Action: Revocation of Missouri license. Effective 8/12/91

Continued on Page 216

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DISCIPLINARY ACTIONS

Continued from page 215

■ **Edward A. Porter, M.D.,** Hannibal, MO.
1/29/92 - Retirement agreement signed in lieu of further investigations or proceedings concerning his license to practice.

■ **Debabrata Ray, M.D.,** St. Louis, MO. Prior to licensure in Illinois, the doctor engaged in the unauthorized practice of medicine. The Illinois Board issued Dr. Ray a license under a three (3) year probation.
Violation: Section 334.100.2(8) RSMo
Board Action: Missouri license issued under probationary terms
and conditions until 9/23/92. Effective 8/1/91.

■ **Ernest C. Rose, M.D.,** Charlestown, IL. Hawaii Board suspended Dr. Rose's license for three (3) months for practicing medicine while impaired and for violating the Uniform Controlled Substances Act. Iowa revoked Dr. Rose's license for failure to report Hawaii's action. Kentucky placed Dr. Rose's license on probation for one year for failure to report Iowa's action. Illinois placed Dr. Rose's license on probation for three years for failure to report Iowa's action.
Violation: Section 334.100.2(8) RSMo Supp. 1990
Board Action: Missouri license issued under one (1) year probation with terms and conditions. Effective 7/9/91

■ **William O. Seabaugh, M.D.,** Cape Girardeau, MO.
10/14/91 - Mandatory retirement as part of 2/10/89 probation.

■ **Cesar Villanueva, M.D.,** Kansas City, MO.
Board Action: Termination of probation. Effective 2/19/92.

■ **David White, M.D.,** Lake Ozark, MO. Personal impairment.
Violation: Section 334.100.2(4a)(6)(13)(22) RSMo
Board Action: One (1) week suspension followed by a five (5) year probation. Effective 10/11/91.

■ **David Yahnke, M.D.,** Tulsa, OK. Personal impairment.
Violation: Section 334.100.2(8) RSMo Supp. 1990
Board Action: License issued on three (3) year probation. Effective 12/20/91.

■ **Frederick Zahn, M.D.,** St. Louis, MO. Conviction of a Class C felony of assault in the second degree, the felony of armed criminal action, and the Class D felony of unlawful use of a weapon.
Violation: Section 334.103 RSMo
Board Action: Revocation of Missouri license. Effective 12/12/91.

Clinic will 'link' the indigent patient into the existing health care system

by Dennis Weiser, Managing Editor

Coleen Kivlahan, M.D., medical director with the Missouri Department of Social Services, has some old-fashioned ideas about health care. One that she feels strongly about is that patients - even those without insurance or money - should not be forced to wait for up to four weeks before they can see a physician. "I've seen that happen, and that kind of delay for primary care is unacceptable," she said. To deal with this problem, and others, Dr. Kivlahan will serve as medical director of a new primary care clinic when it opens for business this spring in Columbia, Missouri.

The clinic, the Family Health Care Center, will be staffed by a nurse-practitioner, public health R.N., clerical staff, and physicians with extensive public health experience, including Dr. Kivlahan. It will "link" indigent patients and their families into the community's existing health care system. "We'll provide an access point for people who have lost or who never had access to our medical care system," said Dr. Kivlahan.

Support for the clinic is being provided by Columbia's hospitals: Boone Hospital Center/ Christian Health Services, University Hospital & Clinics, and Columbia Regional Hospital, the Columbia Area United Way, the Boone County Medical Society Auxiliary, the Columbia City Council, State Representative Chris Kelly and State Senator Roger Wilson, and the Department of Social Services.

These institutions and individuals are supporting the program because if it proves to be effective, health care services to the area's under-served population will improve - no small accomplishment in a society that is trying simultaneously to increase access, keep quality high and reduce costs for medical treatment.

The new primary care clinic will earn its salt by reducing the access delay for persons without health insurance, by cutting the inappropriate use of emergency rooms by those same individuals who present cases of otitis media, minor sprains, and upper respiratory tract illnesses, by decreasing overload anxiety for the primary care providers who currently serve this population, and by lessening the factors that tend to block access into the area's health care system.

"I asked for a five-year commitment to fund the program," said Dr. Kivlahan. "But, we will take advantage of any available government funds the patient might be eligible for to help pay our way. We will have a sliding fee schedule for those who can pay, but we will treat patients regardless. It's not enough that we treat the illness - I have to find out if they have any money to pay for a prescription or if they have a refrigerator to keep it in when they get home.

"If the patient is eligible for aid, we'll shift that person into the proper channels for a paying or insured patient. If he or she doesn't have 50 cents to make a minimum payment for a drug dosage, however, we have to include that into our treatment scenario while we're providing that primary care."

As a public service doctor, explains Dr. Kivlahan, she has to be concerned with both providing quality care and how she can help her patients take advantage of that care in realistic terms. "We're talking about treating some patients who may not have a place to sleep or who may not know where their next meal is coming from," she said.

The clinic will be open for appointments and walk-ins every day for half a working day. The nurses will provide the main contact with patients, working under physician protocols, and a physician will be available in the clinic two days each week and by phone daily for consultation. During the second half of each work day, one nurse will remain in the clinic for acute emergency calls and to perform chart work.

The other nurse will make home visits, provide community health education, and assist in community public health activities such as immunization clinics. The after-hours call system will be 24-hour availability by phone with one of the nurses, a physician back-up for the nurse, and a contractual arrangement with the tertiary care hospital for emergency room coverage.

"One of the most exciting elements of this program," Dr. Kilvahan said, "are the 'self-help' educational efforts being provided by some of the other organizations in the community and by our support-

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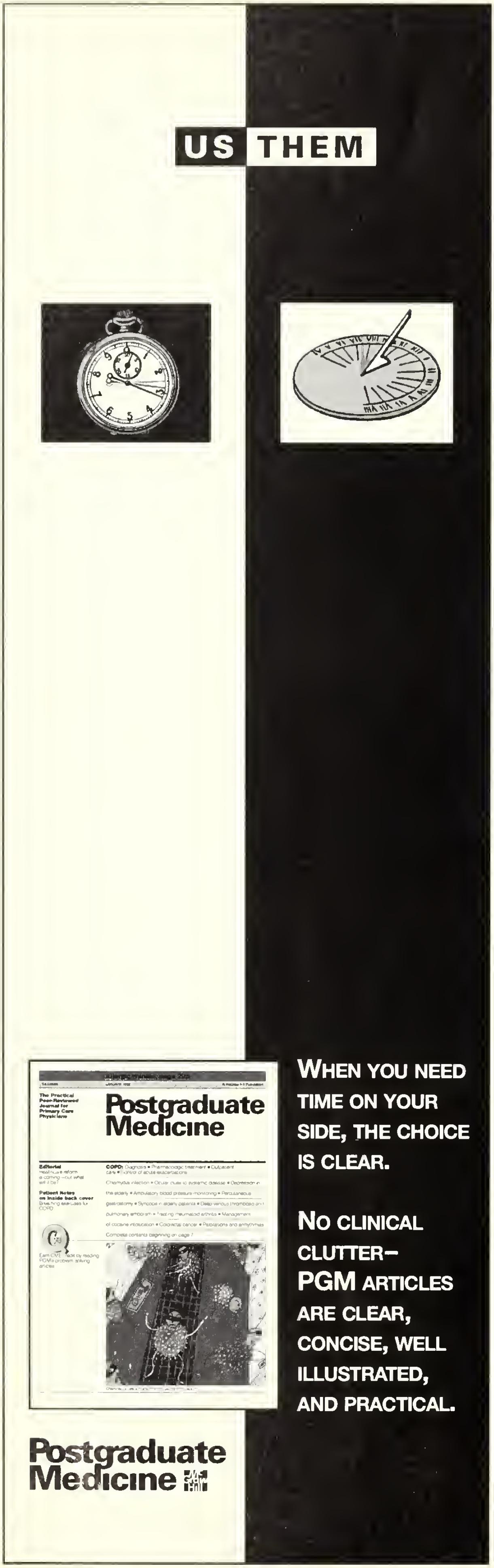
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ers." The Boone County Library, she explains, will emphasize learn-to- read, stay-in-school, get-a-driver's-license, and other such self-help programs in their neighborhood branches.

In addition, the Boone County Medical Society Auxiliary is providing over 40 volunteers who will transport people to the clinic, teach parenting classes, and help with immunization programs. "The Auxiliary is doing a 'fruit of the month' program, too," Dr. Kivlahan said. "The idea may sound a little silly at first, but it is to help people cut back on snack foods by showing what fruits - apples, oranges, bananas - are in season and economical. They will place posters in the clinic that explain the nutritional merits of fruit compared against a typical fast food meal and put out free samples for people to eat."

Self-sufficiency, along with health care access, is Dr. Kivlahan's goal for her patients. She believes that getting people back into the existing health care system is better for them than providing an alternative system.

"Moving off welfare, getting a job, passing a driver's license test - things that most of us take for granted - are major successes for people who live at the lower end of our economic system. Each step up for them is a step away from a social welfare system that tries hard, but which doesn't begin to provide the kind of support and stability that people want for themselves and their families. Success, in terms of health care, means private care. When former welfare recipients get jobs, their first priority is to arrange coverage by a 'real' doctor for themselves and their families."

Defining a success track for patients is one thing, but measuring the success of the entire operation is another, the medical director acknowledges. "Evaluation will be a key element. We will track utilization, especially during the first six months while our client base is building, so we can figure out what we are up against here. We don't have a crystal ball that has told us whom we will be serving, how often our patients will use our services, or what impact we will have in the community. We'll self-correct as we go, as we learn more about what needs to be done and the best ways to go about it".

When indigent families learn the advantages of healthy living habits and gain access into the community's health care system, they can become prudent consumers, said Dr. Kivlahan, but her old-fashioned viewpoint prevents her from exaggerating the long-term effectiveness of a community clinic program.

"It's a temporary fix," she said. "Every family deserves its own family physician." ■

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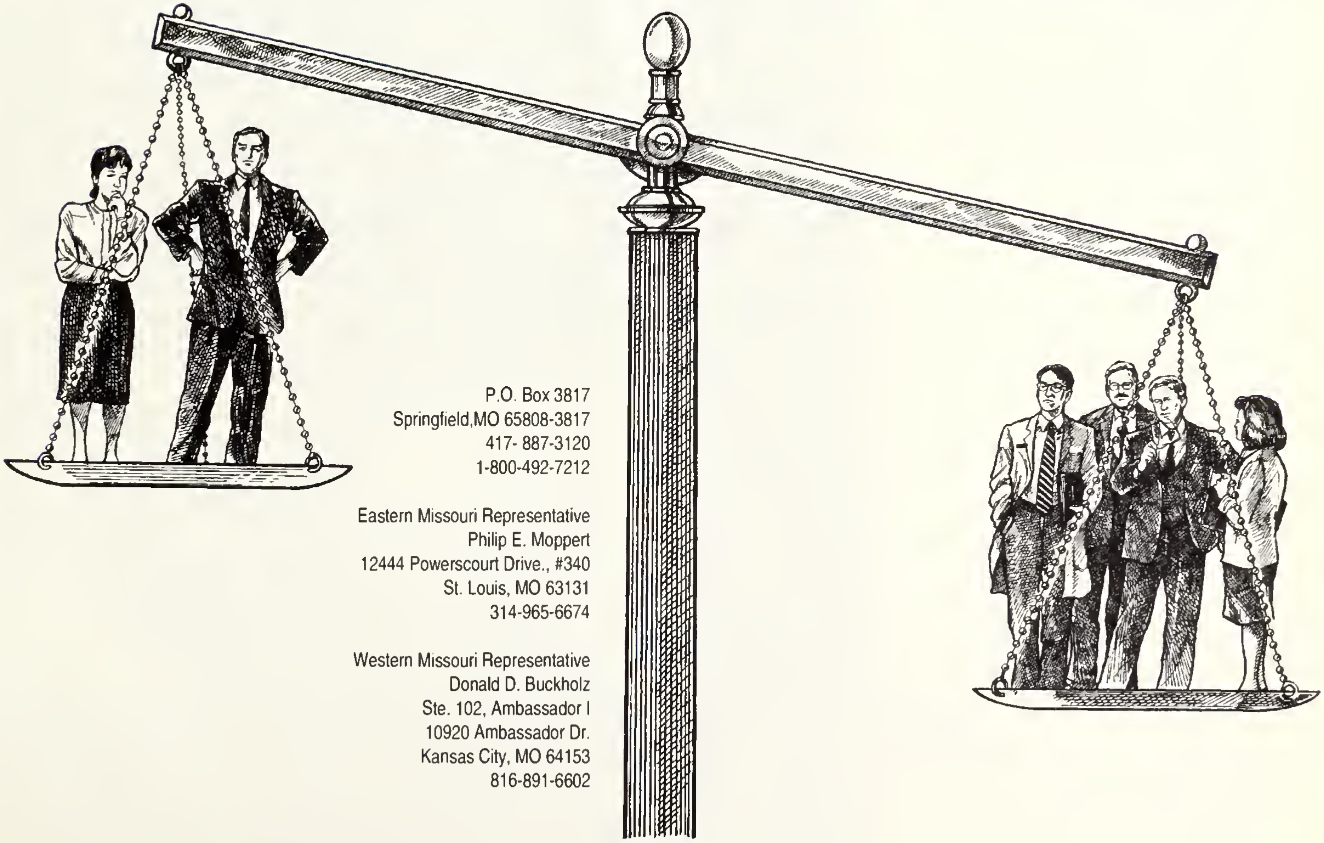

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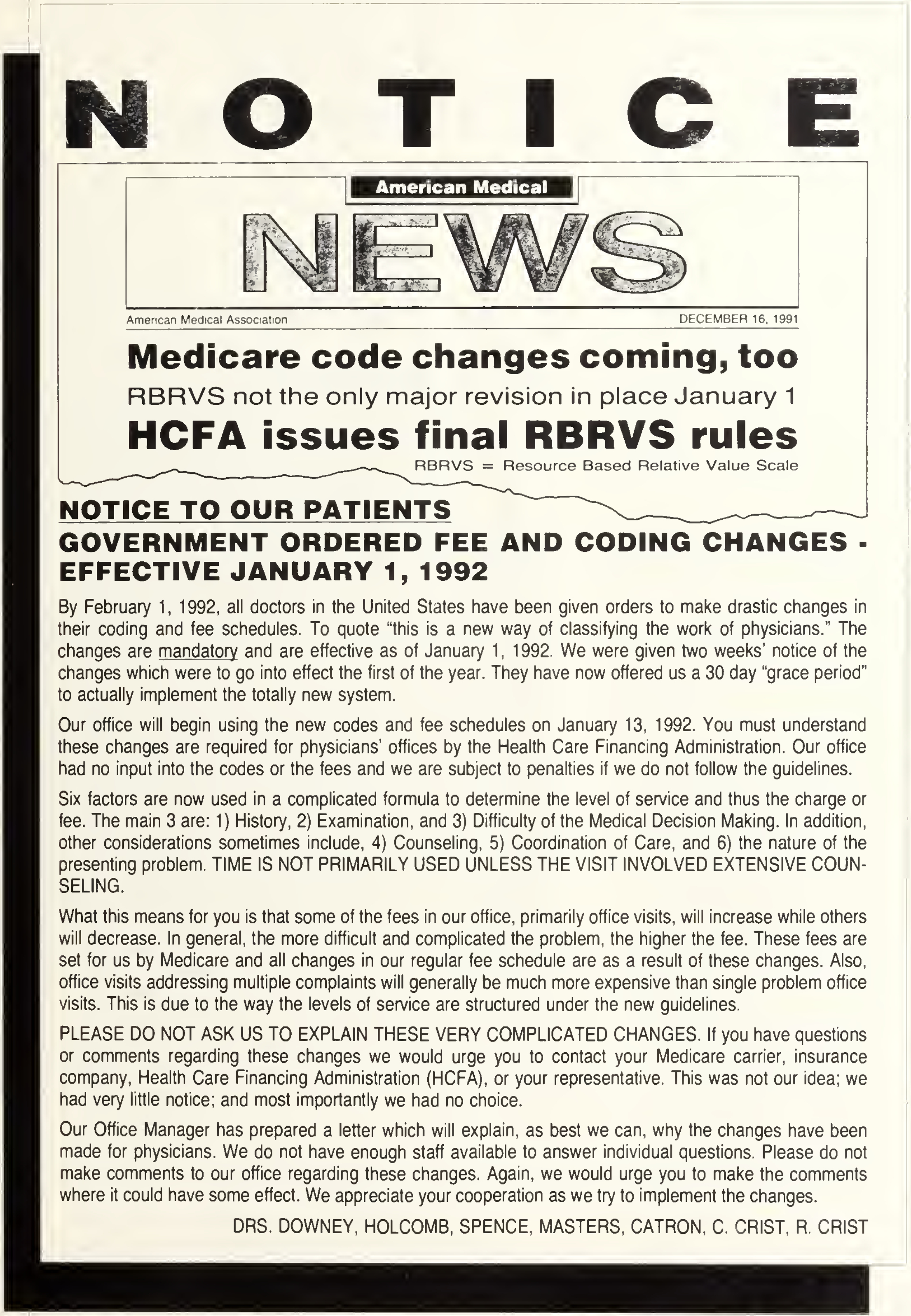

NOTICE

American Medical

NEWS

American Medical Association DECEMBER 16, 1991

Medicare code changes coming, too

RBRVS not the only major revision in place January 1

HCFA issues final RBRVS rules

RBRVS = Resource Based Relative Value Scale

NOTICE TO OUR PATIENTS

GOVERNMENT ORDERED FEE AND CODING CHANGES - EFFECTIVE JANUARY 1, 1992

By February 1, 1992, all doctors in the United States have been given orders to make drastic changes in their coding and fee schedules. To quote "this is a new way of classifying the work of physicians." The changes are mandatory and are effective as of January 1, 1992. We were given two weeks' notice of the changes which were to go into effect the first of the year. They have now offered us a 30 day "grace period" to actually implement the totally new system.

Our office will begin using the new codes and fee schedules on January 13, 1992. You must understand these changes are required for physicians' offices by the Health Care Financing Administration. Our office had no input into the codes or the fees and we are subject to penalties if we do not follow the guidelines.

Six factors are now used in a complicated formula to determine the level of service and thus the charge or fee. The main 3 are: 1) History, 2) Examination, and 3) Difficulty of the Medical Decision Making. In addition, other considerations sometimes include, 4) Counseling, 5) Coordination of Care, and 6) the nature of the presenting problem. TIME IS NOT PRIMARILY USED UNLESS THE VISIT INVOLVED EXTENSIVE COUNSELING.

What this means for you is that some of the fees in our office, primarily office visits, will increase while others will decrease. In general, the more difficult and complicated the problem, the higher the fee. These fees are set for us by Medicare and all changes in our regular fee schedule are as a result of these changes. Also, office visits addressing multiple complaints will generally be much more expensive than single problem office visits. This is due to the way the levels of service are structured under the new guidelines.

PLEASE DO NOT ASK US TO EXPLAIN THESE VERY COMPLICATED CHANGES. If you have questions or comments regarding these changes we would urge you to contact your Medicare carrier, insurance company, Health Care Financing Administration (HCFA), or your representative. This was not our idea; we had very little notice; and most importantly we had no choice.

Our Office Manager has prepared a letter which will explain, as best we can, why the changes have been made for physicians. We do not have enough staff available to answer individual questions. Please do not make comments to our office regarding these changes. Again, we would urge you to make the comments where it could have some effect. We appreciate your cooperation as we try to implement the changes.

DRS. DOWNEY, HOLCOMB, SPENCE, MASTERS, CATRON, C. CRIST, R. CRIST

POSTER HELPS GROUP DIRECT PATIENTS' QUESTIONS ABOUT E/M CODE CHANGES

This poster, original size 18"x24", was presented at the February 2, 1992 Council meeting in Jefferson City, MO. A motion was presented and adopted that it should be published in *Missouri Medicine*. Drs. Downey, Holcomb, Spence, et al, of Cape Girardeau, had these posters printed and display them in their waiting room and examination rooms for their patients to read. Although the effective date for the E\M Code changes was postponed one month, the message remains the same - when the coding system for patient services changes, so will the charges. When such changes occur, patients are invited to address their questions and comments to HCFA and their elected officials in Washington, D.C.

LEGAL CHALLENGES TO DENIALS OF INSURANCE COVERAGE FOR "EXPERIMENTAL" MEDICAL TREATMENTS

By Michael R. Testerman
Springfield, Missouri

Medicare and private health insurers typically deny coverage of so-called "experimental" procedures, devices or drugs. They also will not cover items or services which are deemed "not reasonable and necessary" for diagnosis or treatment of the patient's condition. However, in several important areas, legal conflict is predictable when coverage is denied for these reasons.

Incurable diseases, such as AIDS and some forms of cancer, by definition have no established and effective healing method. Symptoms can be treated, but the underlying disease cannot be cured. In a theoretical sense, every treatment of such diseases is experimental. It is often difficult to determine which measures are experimental within the meaning of the exclusion provisions of insurance policies.

New technology, new drugs, and innovative methods often entail substantial cost. In most instances, patients can afford these efforts only if public or private insurance coverage is available. However, where the measure is deemed "experimental", a denial of coverage usually results. Accordingly, many terminal patients will never receive new treatments for their diseases.

There is a compelling ethical question raised in these situations: Will society simply allow patients to die without attempting new, heroic or innovative measures because there is as yet no clinically proven cure? On the other hand, there is a significant economic question: Will society bear the cost of unproven and often incredibly expensive treatments in the hope that some treatments or combination of treatments may produce satisfactory results in a patient or a particular class of patients?

In many instances, economic cost considerations may lead to denials of coverage, while some patients protest the negative decisions on a ethical basis. As AIDS in particular gains public and political attention, increasing numbers of legal challenges to adverse coverage determinations are forseeable.

WHAT IS "EXPERIMENTAL"?

Medical researchers or other experts may have different standards for determining when a procedure is experimental. They may require a certain number of clinical studies, or acceptances as a routine practice in treating patients, before concluding that the label no longer applies. However, the conflicting opinions of medical experts only compounds the difficulties courts have on the issue. Moreover, economic factors may cause unavailability of new procedures to most patients, and hinder efforts to establish a new treatment as accepted protocol.

Even approval of a product as safe and effective by the U.S. Food and Drug Administration will not necessarily lead insurers or the courts to conclude that it is not experimental. For example, in the case of *Jameson v. Brown*[1], the California court upheld a Medicare carrier's determination that balloon angioplasty was experimental, despite prior FDA approval. It is not universally established in the medical community when some product or technique will cease being deemed experimental. Obviously, FDA approval is not enough.

Webster's Seventh New Collegiate Dictionary defines in relevant part an "experiment" as:

> "1a: Test, trial b: a tenative procedure or policy c: an operation carried out under controlled conditions to discover an unknown effect or law, to test or establish a hypothesis, or to illustrate a known law"

Similarly, *Black's Law Dictionary* defines in relevant part an "experiment" as:

> "A trial or special test or observation made to confirm or disprove something doubtful"

In the absence of a different definition under a particular policy of insurance, common definitions will apply.[2] Thus, it seems that the salient characteristic of an experimental measure is one which has an unknown or doubtful result.

Thus, if a particular treatment is not "known" to produce a cure or satisfactory result for a particular condition, on a theoretical level it may be deemed experimental, and accordingly, not covered by insurance.

PRICING OF NEW MEDICAL TREATMENTS

New medical procedures, drugs or other new items or services often entail substantial cost. One reason for this is the economic dynamics of the health care and pharmaceutical industries.

Research and development often involves tremendous costs to a company or provider. Years of development, testing and regulatory consideration may be involved before a new drug, for instance, can be marketed. The company incurs tremendous costs for researchers, laboratories, studies, attorneys, and the like, before even one sale of the product. A for-profit organization must recover these costs once it is able to market the invention. These costs may be loaded on the product's first few years of existence before some then-unknown alternative item may render the invention obsolete.

On patented drugs or devices, the patent itself has value. The value of the patent right is most significant in the early stages after introduction into the market, and declines as the expiration of the patent grows nearer. Competitive pressures also affect pricing. A patented product is a monopoly, and pricing may be controlled accordingly. Even for non-patented products, if there are no comparable or relatively close substitutes, higher prices are expected.

Price is a function of supply and demand. If the supply of a particular product is limited (such as, interferon), an upward pressure on price results. Upward price pressure also results from limited demand as for a drug used only to treat certain isolated conditions.

The result is that many new, specific or innovative drugs, devices, and treatments are very expensive, thus placing an economic incentitive on insurers to deny coverage so that profit levels or premium costs will not suffer.

COSTS AFFECT CLASSIFICATION AS "EXPERIMENTAL"

There is evidence suggesting that costs affect whether a procedure is deemed experimental by insurers. It is often difficult to quantify the effect costs have on this determination. In some instances, however, the cost of a procedure seems to be the principal characteristic distinguishing noncovered treatments from other treatments which are routinely covered by insurance.

For instance, insurers typically cover the cost of chemotherapy for cancer patients. This is true even for those patients with diseases which, in all probability, will not respond to chemotherapy. For patients with such diseases, it is unrealistic to speak in terms of a "cure." Positive results are obtained even where the spread of the disease is slowed or remission is achieved for some period of time. Certainly, patients respond differently to the same or similar treatment regimen. In other words, chemotherapy may achieve a positive result for one patient while seemingly having no effect on the disease of another.

Oncologists seek the "formula" for positive results in their patients. Different chemotherapy drugs are used in different combinations and dosages, and radiation therapy and surgery and other alternatives are considered as well. In a sense, the oncologist "experiments" with each patient in seeking the combination of treatments to which he or she may respond most positively. However, insurers usually would not deny coverage of these treatments on the basis that they are "experimental."

Some insurers have denied coverage, however, of a procedure known as "autologous bone marrow transplantation" (ABMT) on the basis that it is "experimental." ABMT does not involve a "transplant" in the usual sense of a body part being relocated from a donor to a recipient. Rather, the procedure involves the retrieval and storage of a patient's own bone marrow. Chemotherapy can destroy a patient's bone marrow, thus leaving him more susceptible to life-threatening complications. With ABMT, marrow is kept outside the body while higher than usual chemotherapy dosages are administered. After the prescribed chemotherapy sessions, the patient's own bone marrow is reinjected and, thus, is not so severely damaged by the drugs.

In *Thomas v. Gulf Health Plan, Inc.*,[3] after precertification of paying costs for the ABMT procedure on a breast cancer patient, and after the bone marrow had been "harvested" from the patient, the insurer denied coverage on the grounds that the treatment was experimental. The court found, based on medical opinions presented to it, that although ABMT was not experimental for treating several types of cancer, it **was** experimental as a breast cancer treatment. Thus, the court upheld the denial of insurance coverage to that patient.

Why the ABMT deemed any more "experimental" than traditional chemotherapy? The distinction in the chemotherapy used on ABMT patients versus other cancer patients may be in the **dosage** of the drugs used. If chemotherapy is not experimental for most patients, why would higher doses of the same or similar drugs necessarily be experimental? It seems that dosage does not require such a finding since dosage typically varies from patient to patient. Is the removal, storage and reimplantation of a patient's own bone marrow an "experimental" procedure? Again, nothing in this process seems particularly unique or experimental. However, bone marrow harvesting and reinjection combined with increased chemotherapy doses leads some insurers to the conclusion that the overall treatment is experimental.

Is this determination a function of the fact that some ABMT patients do not achieve a cure or remission? No, it seems traditional chemotherapy may produce even less satisfactory results. Is it the fact that there are increased risks to ABMT patients due to, among other things, toxicity of the drugs or temporary depletion of the marrow? It seems not, since traditional chemotherapy carries risks, as do all other treatment alternatives such as radiation or surgery. Does relative newness of a technique require a finding that it is experimental? That involves a subjective evaluation of how new is new. Where studies demonstrate that encouraging results have been achieved,[4] is the method any more experimental than the alternatives? In searching for the logical reason to the cost factor. The cost of ABMT can exceed $100,000, since the patient often faces substantial in-patient days and relatively few facilities offer the treatment.

Perhaps the exlcusion of experimental treatments is a veiled method for protecting insurers against the cost of new methods or technologies. An insurer (subject to monetary coverage limits) cannot deny coverage simply due to cost so long as the cost is in line with usual and customary charges. However, since costs of newer procedures are usually higher than costs of more traditional approaches, an exclusion of experimental treatments serves as a *de facto* cost containment measure.

LEGAL CHALLENGES TO THE DENIAL OF COVERAGE FOR "EXPERIMENTAL" PROCEDURES

In Missouri, legal challenges to insurers' denial of coverage for experimental treatments face difficult obstacles. One basis for such a challenge would center on Missouri's statutes (R.S. Mo. 375.296 and 375.420) which provide penalties for an insurer's "vexatious" refusal to provide coverage.[5] Only by demonstrating some bad motive may a patient have a possibility of winning a case under these laws.

For new drugs, treatments, devices, etc., this may prove to be an insurmountable task. Missouri law provides penalties only where the denial of coverage is both "vexatious" and "without reasonable cause."[6] If an insurer has reasonable cause to believe that there is no coverage under its health insurance policy, it is not subject to the penalties provided under these statutes.[7] Moreover, where there is a "genuine dispute" for the insurer to require adjudication, the insurer will not be penalized for denying coverage pending court determinations.[8]

For many new treatments, an insurer will have little difficulty obtaining a physician's opinion that they are experimental. Medical literature and reports of studies may also refer to the procedures as experimental or investigatory. It is difficult to determine when new procedures stop being experimental. Perhaps this only comes after years of study and, ultimately, routine acceptance by the medical community. Until such point, it seems that an insurer would have some "reasonable cause" to believe that treatments are experimental and thus not covered by its insurance policy.

There are also practical obstacles to legal challenges by some patients. A patient suffering from AIDS or advanced forms of cancer may need to either receive the new treatment immediately or never at all. A patient who fits the "profile" for receiving such treatments may deteriorate and no longer be a candidate for the treatment after the passage of some time. Unfortunately, after making demands for coverage, awaiting a response, contacting an attorney, retaining expert witnesses, and preparing a case for eventual determination in court, it may be too late for that particular patient. This barrier can be lessened somewhat by suing for an injunction against the denial of coverage, rather than initially proceeding under the punitive statutes discussed above.

A temporary restraining order or preliminary injunction could require a company to provide benefits, and may be sought very soon after suit is filed. The person seeking such relief must show: (1) Irreparable harm will occur without an injunction; (2) The harm to the plaintiff if the injunction does not issue outweighs the harm to the defendant if it does; (3) It is probable that the plaintiff ultimately will succeed on the merits of the claim; and (4) The interests of the public favor the granting of the injunction or, at minimum, do not unduly weigh against it.[9]

The largest obstacle in this context is whether it is likely that the plaintiff will ultimately prevail on the merits of the claim that the particular medical procedure, device, or whatever, is covered by insurance and is not experimental. This determination may require expert medical opinions, medical studies and other proof which can be expensive and time consuming to gather.

Courts will generally enforce the language of an insurance policy if it is unambiguous. Thus, if a policy excludes coverage of experimental procedures, courts will enforce that exclusion if it is clearly and unequivocally written. All that is left to decide is whether a particular treatment is "experimental" within the meaning of that particular insurance policey. If the policy provisions are ambiguous, the policy will be construed in the patient's favor.

In the case of *DiDomenico v. Employers Cooperative Industry Trust*,[10] the health insurance policy provided that it did not cover "transplants which are considered experimental or are done for purposes of research. Transplants of a kidney, cornea or a liver (for children under age 12) are not considered experimental." Mr. DiDomenico, an adult, was diagnosed as having biliary cirrhosis of the liver. Doctors at the University of Chicago determined that he should receive a liver transplant and that his life was in grave danger without the procedure. The insurer denied coverage, stating that the treatment was experimental.

The *DiDomenico* court found "the language of the plan relating to organ transplants is, to engage in understatement, ambiguous." Thus, the policy language was strictly construed against the insurance company. The court granted plaintiff's request for a preliminary injunction against the denial of coverage, thus allowing Mr. Domenico to receive the transplant.

No doubt, there have been many situations where insurance coverage is denied on the basis that the treatment sought is "experimental". However, the patient must be able to muster the will and financial resources to challenge the insurer's determination. Undoubtedly, many patients with weakened health and already strained pocketbooks simply accept the insurance company's position as yet another cruel turn of life.

CONCLUSION

Patients with "incurable" diseases hang onto strained threads of hope. It is a sad situation when those threads are severed for the sake of insurance company profits. However, we must recognize that costs covered by insurance are recouped through premiums, some new techniques simply cannot be made available to everyone. As researchers search for new treatments for AIDS patients and others, society will necessarily wrestle with the reality that many innovative treatments may be available only to the very wealthy. Some conflicts between terminal patients and their health insurers will be decided in the courts. The decision of judges and juries througout the country will steer the insurers and may determine the ultimate fate of thousands of desperate patients.

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[5] Missouri Revised Statute 375.296 provides:

In any action, suit or other proceeding instituted against any insurance company, association or other insurer upon any contract of insurance issued or delivered in this state to a resident of this state, or to a corporation incorporated in or authorized to do business in this state, if the insurer has failed or refused for a period of thirty days after due demand therefor prior to the institution of the action, suit or proceeding, to make payment under and accordance with the terms and provisions of the contract of insurance, and it shall appear from the evidence that the refusal was vexatious and without reasonable cause, the court or jury may, in addition to the amount due under the provisions of the contract of insurance and interest thereon, allow the plaintiff damages for vexatious refusal to pay any attorney's fees as provided in Section 375.420. Failure of an insurer to appear and defend any action, suit or other proceeding shall be deemed prima facie evidence that its failure to make payment was vexatious without reasonable cause.

Missouri Revised Statute 375.420 provides:

In any action against any insurance company to recover the amount of any loss under a policy of fire, cyclone, lightning, life, health, accident, employer's liability, burglary, theft, embezzlement, fidelity, indemnity, marine or other insurance, if it appears from the evidence that such company has vexatiously refused to pay such loss, the court or jury may, in addition to the amount thereof and interest, allow the plaintiff damages not to exceed ten percent on the amount of the loss and a reasonable attorney's fee; and the court shall enter judgement for the aggregate sum found in the verdict.

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LIABILITY EPIDEMIC - HEALTH CARE COSTS:
Break the Linkage

By Charles C. Norland, M.D.

Are the politicians finally ready to take the most critical step needed to start control of medical costs? The medical community knows that medical costs will not come down in any meaningful way until the liability epidemic is controlled. The politicians are debating endless complex ways of regulating medicine. As of January 1992 they have physicians under rigid federal wage and price controls; the rush to add more control continues like a feeding frenzy. There is, however, a bright spot because there is well drafted civil justice reform legislation working through Congress which, if passed, will alter the practice of medicine in a very positive way.

Why are the politicians studying the Canadian provincial-government health insurance model and the German social insurance model? The answer is obvious - they provide universal coverage and they spend less than the United States. They spend less because they have a much lower cost of administration, they provide less care (Canada) and they don't have our legal system. Legal reform is absolutely necessary as a first step to cut costs in medical care because to give us German type medical care without giving us German type common sense law would be a cruel hoax. We would impose great change and then have care which costs more than our present system. We are debating the cart and it is way out in front of the horse.

We need judicial reform as outlined in "A Report from the President's Council on Competitiveness - Agenda for Civil Justice Reform in America" published in August 1991.[1] This report, which deserves careful study, states

> **Many features of the current legal system no longer serve to expedite justice or to ensure fair results. Instead, overuse and abuse of the legal system imposes tremendous costs upon American society. Each year the United States spends an estimated 300 billion dollars as an indirect cost of the civil justice system.**

Walter K. Olson in a recent article in *Medical Economics* states, "America is the only major country that denies the winners of a lawsuit the right to force the loser to pay his legal expenses. In other countries the promise of a fee recoupment from the opponent gives lawyers good reason to take on a meritorious case for even a poor client."[2] To assure that poor people have access to the courts, judicial discretion could waive fee recoupment for the poor when appropriate.

Civil law reform should have been one of the first legislative actions taken to control medical costs. Congressional fear of the vocal, organized, and well-financed trial lawyers and the consumer advocates must be the major factor. Recently, I discussed this with Congressman Richard Gephardt and he agreed that judicial reform was needed and could be passed by Congress in spite of the fact that lawyers predominate as members of Congress. Grassroots support from businessmen, engineers, carpenters, farmers and people from all walks of life is self evident. The abstainers would be the trial lawyers and the consumer advocates and these two are inseparable.

America can't compete in the world with our present system of civil law. We have 70 percent of the planet Earth's lawyers in the United States yet we only have five percent of the world's population. Japan has one lawyer for every 10,000 people while the United States has one for every 250 people. The cost to business for product liability is 15-20 times (1,500 - 2,000 percent) higher in the United States than in Europe or Japan. The medical profession spends 25 billion dollars a year on malpractice premiums. The AMA estimates that defensive medicine adds 21 billion to the United States health care bill every year. Other experts like former U.S. Surgeon General C. Everett Koop believe the cost is three times that high or 63 billion dollars. Whatever the amount, most of it should be directed to meeting America's medical needs.

Facts should be the primary concern in the courts that make us swear to tell the truth, the whole truth and nothing but the truth. Between 1923 and the mid 1970s the Frye rule was operational in the courts. Testimony by an expert witness would only be admitted if it had gained general acceptance in the particular field. Since the early 1970s the courts have essentially done away with this rule. Peter Huber, in an article "Junk Science in the Courtroom" states,

> **An expert who appears in court to present nothing but his own idiosyncratic opinions is, for all practical purpose, just a lawyer in scientific drag. Science, by definition, is never a matter of individual opinion; it is always a matter of consensus in a much larger community.**[3]

Testimony on medical practice and science has to be based on research which is published and presented to the scientific community for peer review. Confirmation by other scientists and discussion in the scientific community gives us the generally accepted consensus. The courts can find consensus if they want, but judge and the laws have to want the truth, the whole truth and nothing but the truth.

Most medical malpractice suits have nothing to do with negligence. A recent study by Harvard researchers that appeared in a 1991 edition of the New England Journal of Medicine showed that in four of five lawsuits filed there was no negligence.[4] The major ingredient in a medical malpractice suit is a patient with major body damage who is litigious.

The classic example is cerebral palsy. There are about four million births a year in the United States and some 4,000 of these children have cerebral palsy. Ten percent of natural deliveries are categorized as difficult deliveries. Most of the 400 cerebral palsy cases with a difficult delivery came to trial. Many times each day ads on television attempt to lure these cases into court. Huber states, "The junk science theory here is that obstetrical malpractice is an important cause of cerebral palsy, that better use of electronic fetal monitoring and more aggressive use of the scalpel for Cesarean delivery would often prevent this affliction."[3] The scientific evidence that physicians use to make medical decisions, as reported in a 1986 New England Journal of Medicine article, shows "the risk of cerebral palsy associated with each factor related to labor and delivery is under two percent."[5]

For the last three decades these cases of cerebral palsy have continued to come to trial in multiple scattered courtrooms around the United States with much the same script. First the jury is told about the undisputed difficult delivery. Next the junk scientist quoting unsubstantiated opinion and a few out-of-date studies testifies for the plaintiff. This is countered by expert testimony for the defendant quoting in-depth studies covering thousands of cases showing no meaningful connection. Lastly, the jury sees the incontinent child in a wheelchair. The Cesarean section rate in the United States has increased from five to 25 percent with increased risk to the mother and absolutely no change in the incidence of cerebral

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Excerpts from **A Report from the President's Council on Competitiveness**

Agenda for Civil Justice Reform in America

Voluntary Dispute Resolution

Most potential litigants consider only two avenues for dispute resolution: informal negotiation and litigation. The Council on Competitiveness recommends providing greater access to alternative dispute resolution (ADR) techniques that would routinely be available as a substitute for traditional litigation. These techniques include (1) early neutral evaluation, (2) mediation, (3) arbitration, and (4) summary jury trials. The Council also encourages the private sector to employ contract provisions establishing non-judicial means of dispute resolution in order to introduce ADR into the corporate process.

Because more than 92 percent of all civil lawsuits are settled or otherwise disposed of prior to trial, the Council encourages mandatory settlement conferences soon after the commencement of any litigation. The Council suggests that litigants be required to notify the opposing parties of their intention to file suit before resorting to the courts. These actions will enhance the possibility that disputes may be resolved amicably without the courts.

palsy. If the lawyers and the junk scientists were right, we would have seen a decreased incidence of cerebral palsy. In spite of this indisputable evidence, the cases just keep going and going to court. Whatever happened to legal precedent?

The plaintiffs's lawyers are so taken by the junk scientists that it is unusual to have a legitimate scientific witness testify for the plaintiff. The words of the plaintiff's expert witness are like listening to a dramatic fantasy from the tooth fairy. It is usually opinion based on no facts, or facts out of context or facts from another era. A former president of the American Bar Association is quoted in a book by Walter Olson as saying, "I would go into a lawsuit with an objective, uncommitted, independent expert about as willingly as I would occupy a foxhole with a couple of noncombatant soldiers."[6] A federal judge, also quoted in Olson's book, once stated, "An expert witness can be found to testify to the truth on any factual matter no matter how frivolous."[6] The law gives equal weight to the astrologist (junk scientists) and the astronomer (scientific fact) with only the safety net of a lay jury to distinguish between the two and the jury gets no help from the courts. The courts just let the Frye rule slip into oblivion.

When junk scientists rule the courts, physicians live in fear. It affects everything we do: the way we order tests, make referrals and document in the chart. We would be fools under the present laws not to practice expensive defensive medicine. We practice with a gun at our head. Should we be motivated by fear or truth? Medicine can live with truth but society can't pay for the consequences of the fear we live with every day. Medicine is a component of the GNP equation while legal expense is not even figured into the GNP equation, but lawyers readjust wealth within the GNP. It is time to do away with the invisible fist of fear because society can't pay for it and it adds nothing to the goals of society.

With this background, how can we bring about change? I strongly encourage you to get a copy of the Report from the President's Council on Competitiveness - Agenda for Judicial Reform in America and study its recommendations. The Council has recommended fifty specific changes to our current civil rights justice system. Of particular interest to medicine are the following:

- Require expert testimony to be based on "widely accepted" theories. A party would have to prove that its expert opinion is based on established theory supported by a significant portion of experts in the field.
- Contingency fees for experts be eliminated.
- A loser pay rule would be applicable in most cases. This is grounded on the English Rule in the equitable principal that a party who suffers should be made whole.
- Punitive damages would be awarded in a consistent manner as part of a coherent system. The amount of the damages should not exceed the full amount of the compensatory damages.
- Reforms in the pre-trial discovery process.
- Promote voluntary use of alternate dispute resolution techniques.

Continued on page 230

Discovery

Pretrial discovery is frequently the source of needless delay and expense. Currently litigants have virtually unlimited ability to take sworn depositions of witnesses, request documents and submit written questions to parties.

The Council recommends several fundamental reforms to the discovery process, including disclosure of basic information and an initial round of discovery that would continue to be "free" to the requesting party. This would include an extensive document request, a limited set of depositions and written questions. Beyond this initial round, however, the requesting party would have to pay for additional discovery. The Council also recommends that greater sanctions be imposed if materials are wrongfully withheld.

More Efficient Trials

Litigation is sometimes necessary when the parties are unable to resolve a dispute through less formal means. The Council recommends several reforms designed to make trial practice more efficient, including urging courts to set early trial dates and to employ summary judgment procedures more frequently to eliminate disputes that can be properly resolved by legal ruling alone.

Expert Witness Reform

Reform of expert witness practice is also essential if trials are to remain a fair and rational mechanism for conflict resolution. One of the Council's principal recommendations in this regard is to require expert testimony to be based on "widely accepted" theories. This would eliminate testimony unsupported by professional practice or

scientific knowledge. The Council also recommends banning contingency fees for expert witnesses. This should prevent expert witnesses from becoming mercenaries or advocates, instead of impartial and objective witnesses.

Punitive Damages

The current common law approach to punitive damages frequently distributes awards in a random and capricious manner. While some states have attempted limited reform, the Council recommends a comprehensive package designed to limit and restrict punitive damages.

The Council recommends that punitive damages be awarded in a rational and consistent manner as part of a coherent system. Plaintiffs seeking punitive damages should not be allowed to assign a specific dollar amount to their requests. Where there is clear and convincing evidence of the defendant's intent to cause injury, then punitive dames may be awarded in a separate phase of the proceeding. In any event, however, the amount of punitive damages should not exceed the plaintiff's actual damages.

Improved Use of Judicial Resources

The Council encourages courts to employ more efficient case management techniques, including flexible assignments of federal district judges. The Council also encourages greater emphasis on resolving intracircuit conflicts. The council has proposed that circuit courts be maintained at a manageable number of judges, and that the Ninth Circuit, which has almost thirty sitting judges, be split into two circuits.

Enhanced Incentives for Reduced Litigation

In order to promote more disciplined and less wasteful litigation, The Council proposes to strengthen the current rules which authorize judges to sanction attorneys who file frivolous lawsuits.

The Council also recommends a test of a modified two-way fee-shifting arrangement whereby the loser of a lawsuit pays the costs incurred by the winner. Although limitations would be built into this system to safeguard equal access to the courts, this reform would encourage pretrial settlements and impose market discipline on the litigation process.

The Council further proposes a moratorium on the more than 150 one-way fee-shifting statutes under which victorious plaintiffs recover their fees from losing defendants, while victorious defendants get no such recovery.

Reducing Unnecessary Burdens on Federal Courts

The federal district and appellate caseloads have skyrocketed in the past decade, creating endless delays and adding substantially to the cost of litigation. The Council has proposed several reforms designed to unclog the federal courts, including revising the threshold for federal diversity jurisdiction, restoring judicial immunity to state court judges for official actions, and reducing abuse of habeas corpus petitions.

Eliminating Litigation Caused by Poorly Drafted Legislation

The Council recognizes that the federal government bears a great deal of responsibility for the rise in litigation caused by poorly drafted federal statutes. The Council proposes that the Executive Branch closely review all legislative proposals against a "litigation hazards" checklist.

Council Recommendations: Implementation

The civil justice reforms proposed by the Council may be implemented through one of several approaches. The five principal methods of implementation are (1) federal legislation, (2) revisions to the federal rules of civil procedure and evidence, (3) judicial action, (4) model state legislation and rules, and (5) executive action. Some of the proposed reforms can be implemented through more than one approach. Changes to the discovery process, for example, may be accomplished either by federal statute or through rule changes. Most reforms are also suited to model state statutes, particularly because they permit reforms suggested for the federal systems to be applied in the state courts.

The Administration will draft legislation to implement these recommendations and will work with Congress toward reform. Other changes will require amendment to the Federal Rules of Civil Procedure, the Federal Rules of Evidence, and the

Federal Rules of Appellate Procedure. The Administration will draft proposed rules changes and submit the proposals to the Supreme Court's Rules Advisory Committees.

Some reforms can also be accomplished locally by the efforts of individual judges. The Administration will make available innovative management procedures and provide technical assistance to support these suggestions.

Although the reforms contemplate Federal law and rules changes, many of the reforms may be equally applicable to state court systems. The Administration will draft model legislation and model rules changes so that these reforms may be implemented at the state level.

The Administration is committed to the fair, efficient, and early resolution of disputes. To underscore this commitment, the Administration will apply many of the suggested reforms to the conduct of litigation by federal agencies.

Note: for your copy of A Report from the President's Council on Competitiveness: Agenda for Civil Justice Reform in America, please call the office of the Vice President of the United States at 202/456-6222, or White House Publications at 202/395-7332

Passage of these measures will take the gun away from the physician's head and allow us to stop practicing defensive medicine. The cost savings will be significant. Meritorious malpractice will not be eliminated. The application of truth to malpractice cases will enhance the law and the public's perception of the law.

In the December 1991 issue of *St. Louis Metropolitan Medicine*, Arthur H. Gale did a Medical Liability Survey which concluded that St. Louis physicians "have very little confidence in, and respect for, our present legal institutions in the area of tort liability."[7] As a delegate to the Missouri State Medical Association's Annual Meeting, I will propose a similar survey be sent out to all physicians in Missouri and include questions related to the Agenda for Civil Justice Reform in America. If this survey confirms the St. Louis findings, we have to broadcast our results and resist changes in health care delivery until we have civil justice reform.

As always, knowledge is necessary to make meaningful change in the scientific community. Walter K. Olson has an article entitled "Can We Ever Halt America's Litigation Epidemic?"[2] and a book *The Litigation Explosion*.[6] Peter Huber has an article "Junk Science in the Courtroom"[3] and a book *Galileo's Revenge - Junk Science in the Courtroom*.[8] Careful study of these two authors gives one a solid background to understand the civil justice reform needed in the United States.

The numbers of the opposition to this legislation will be small but they will be vocal, organized and well-financed. Well-placed TV ads telling Americans that "their key to the courthouse will be taken away" constitute powerful and inflammatory statements but they are false statements. Widows, orphans, and the poor will have their legitimate day in court. Lawyers can still accept them as clients on a contingency basis and if they lose, the courts will have the option to waive fee recoupment. Lawyers for the plaintiff, however, will not have the right to do harm to the people they litigate against if they are found not guilty because the loser pay rule will be applicable. The loser in a civil lawsuit will have to pay the court costs and the lawyer's fees of the winner. This is the rule in the other major countries of the world.

We must all work for bipartisan support of this civil justice reform. The U.S. citizens in general and physicians in particular have lost respect and confidence in our civil justice legal system. This loss of confidence and respect plus the tremendous cost will lead to reform. What better time is there than now to make the change.

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DIAGNOSIS AND MANAGEMENT OF ULNAR NERVE ENTRAPMENT

Cesar A. Gumucio, M.D., Herluf Lund, M.D., V. Leroy Young, M.D., and Ann E. Young

Ulnar nerve entrapment can be divided into compression at the elbow and the wrist. Etiologies range from direct trauma to metabolic derangements. Guidelines for diagnosis at each site have been presented. Surgical release at the elbow begin at the mid arm where the nerve passes from the anterior to the posterior compartment through the intermuscular septum and extends into the proximal forearm, where it dives down under the heads of the flexor carpi ulnaris. Surgical release at the wrist involves deroofing the canal of Guyon. The authors' preferred methods of release at each site are given along with a description of positive care.

Common sites of compression of the ulnar nerve are at the elbow and wrist. The anatomy of the ulnar nerve in these two locations produces specific syndromes which aid in establishing a diagnosis and in localizing the site of nerve compression. Prompt diagnosis and early compression minimize damage to the nerve and resultant disability. The purpose of this article is to discuss the anatomy, diagnosis and treatment of ulnar nerve compression.

Ulnar Nerve Entrapment at the Elbow

Etiology

Potential causes of ulnar nerve entrapment at the elbow include arthritis, trauma, congenital deformities, external compression, metabolic problems, occupational trauma, and tumors. Traumatic causes include fractures of the medial humeral epicondyle and supracondylar fractures, dislocation of the elbow and direct contusion. Rheumatoid and degenerative arthritis are the most common types of arthritis to produce ulnar nerve compression at the elbow. Congenital causes include enlargement of the medial head of the triceps muscle, epitrochleoanconeus muscle, cubitus valgus, subluxing ulnar nerve, arcade of Struthers, and medial head of the flexor carpi ulnaris.[1,2,3] Systemic diseases commonly associated with symptoms of nerve compression include hypothyroidism, uremia, diabetes and alcoholism. Occupations associated with ulnar nerve symptoms include any tasks which requires repetitive heavy lifting or exposure to vibration. Truck drives who rest their arm on the window or door rest of their truck are also at risk. Lipoma and ganglions are the most common tumors to produce ulnar nerve compression at the elbow.

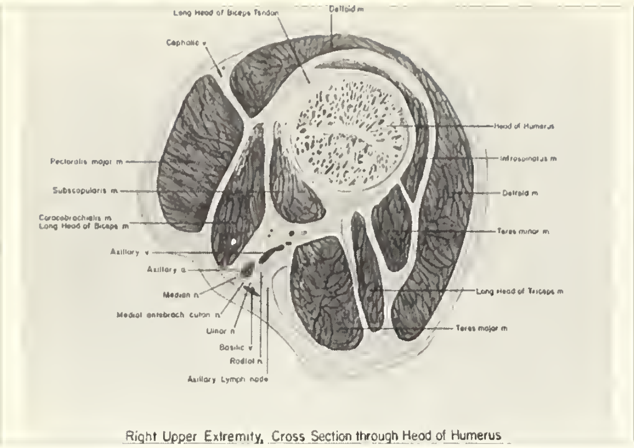
Pertinent Anatomy[4]

The ulnar nerve arises from the cervical root C8 as a terminal branch of the medial cord of the brachial plexus. In the brachium (or arm) it lies within the vascular sheath medial to the brachial artery (Figures 1A and 1B). At roughly the mid portion of the arm, the ulnar nerve passes posteriorly through the intramuscular septum to run along the anterior border of the triceps muscle to the cubital tunnel. In the distal arm the nerve is just beneath the muscular fascia and is easily visualized with minimal risk of injury during surgery (Figure 1C). At the elbow the nerve enters the cubital tunnel and passes between the medial epicondyle of the humerus and the olecranon (Figure 1D). Distal to the elbow the nerve exits the cubital tunnel and courses through the flexor carpi ulnaris muscle (Figure 1E). In the proximal forearm the nerve lies on the anterior surface of the flexor digitorum profundus muscle mass (Figure 1F).

Diagnosis

The diagnosis of ulnar nerve entrapment at the elbow is established by the typical history, findings on physical examination, and nerve conduction studies. Radiographs and laboratory test are of assistance in ruling out fractures, hypothyroidism, arthritis, diabetes and alcoholism.

The typical history of a person with ulnar nerve compression at the elbow can include numbness in the


Long Head of Biceps Tendon
Deltoid m
Cephalic v
Head of Humerus
Pectoralis major m
Infraspinatus m
Subscapularis m
Deltoid m
Coracobrachialis m
Long Head of Biceps m
Teres minor m
Axillary v
Axillary a
Median n
Long Head of Triceps m
Medial antebrach cutan n
Ulnar n
Teres major m
Basilic v
Radial n
Axillary Lymph node

Right Upper Extremity, Cross Section through Head of Humerus

Figure 1A: Right upper extremity cross section through head of humerus demonstrating the relationship of the ulnar nerve to the axillary vessels and the median and radial nerves.

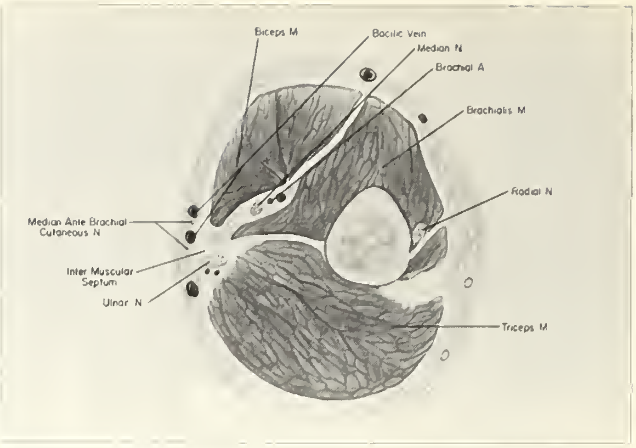
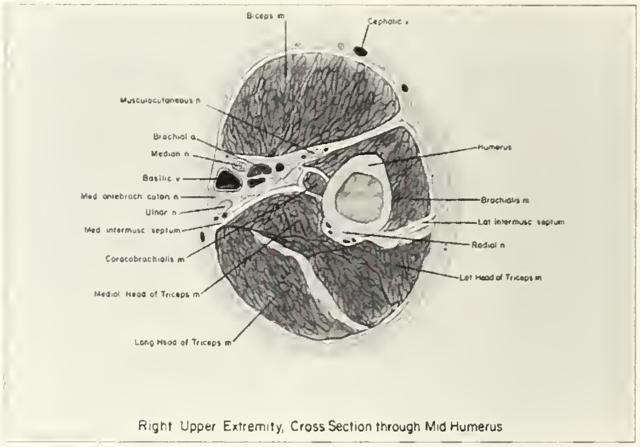

Biceps M
Bacilic Vein
Median N
Brachial A
Brachialis M
Radial N
Median Ante Brachial Cutaneous N
Inter Muscular Septum
Ulnar N
Triceps M

Figure 1B: Right upper extremity cross section through the mid humerus demonstrating the relationship of the ulnar nerve to the basilic vein, median antebrachial cutaneous nerve, intermuscular septum, median nerve, and brachial vessels. The ulnar nerve is anterior to the intermuscular septum and adjacent to the basilic vein and median antebrachial cutaneous nerve.


Biceps m
Cephalic v
Musculocutaneous n
Brachial a
Median n
Humerus
Basilic v
Med antebrach cutan n
Brachialis m
Ulnar n
Lat intermusc septum
Med intermusc septum
Radial n
Coracobrachialis m
Lat Head of Triceps m
Medial Head of Triceps m
Long Head of Triceps m

Right Upper Extremity, Cross Section through Mid Humerus

Figure 1C: Right upper extremity cross section through distal humerus. The ulnar nerve lies posterior to the intermuscular septum on the triceps muscle. The basilic vein and median antebrachial cutaneous nerve are in the subcutaneous tissues. This is the safest and easiest location in which to find the ulnar nerve.

little and ulnar half of the ring fingers or the dorsum of the little and ring fingers; pain in the forearm, elbow or arm; and tenderness or hypersensitivity around the elbow.[5]

The physical findings depend on the exact site, extent and duration of the compression. In early compression there may be tenderness over the nerve at the site of compression, a positive Tinel's sign and a positive elbow flexion test. The elbow flexion test is performed by having the patient forcefully flex the elbow and hold it in that position for one minute. If numbness or paresthesia develop within that minute, the test is positive. In more advanced cases of compression, there is often wasting of the ulnar-innervated intrinsic hand muscles. This is most pronounced in the first dorsal interosseous muscle and in the muscles of the hypothenar eminence.

Nerve conduction studies[6,7] typically show a decrease in conduction velocity across the elbow. Conduction proximal to and distal to the site of compression should be normal. False positive nerve conduction studies showing slowed conduction in the ulnar nerve across the elbow are relatively common. In general, slowed conduction velocities within 25% of a laboratory's normal values are probably not significant. In contrast, a reduction in velocity of 33% or greater is significant.[8] When evaluating a patient for any compressive neuropathy in the upper extremity, both extremities should be tested for conduction in the ulnar and median nerves across the elbow and at the wrist. This establishes an internal control an helps detect polyneuropathies and resolve questions about symptoms in multiple nerve distributions. Abnormal nerve conduction in the absence of symptoms or physical findings should be viewed with suspicion. Ultimately the clinical scenario must provide the guidance for decision making. When the diagnosis is questionable, the patient should be followed with repeat examinations or referred to a neurologist for a second opinion.

Conservative Treatment

Conservative treatment of ulnar nerve compression at the elbow can relieve symptoms. Usually this requires relief of an offending activity or treatment following

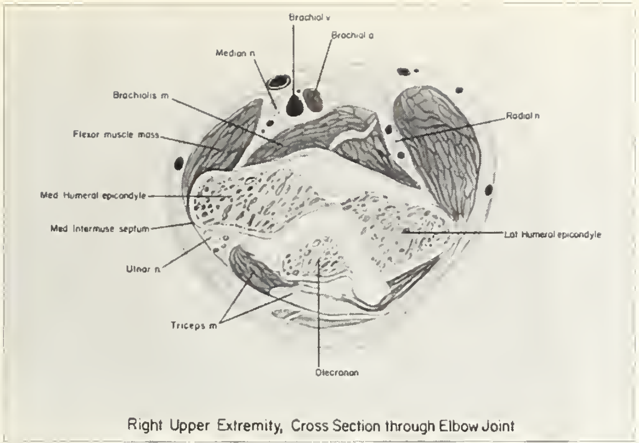

Brachial v
Brachial a
Median n
Brachialis m
Flexor muscle mass
Radial n
Med Humeral epicondyle
Med Intermusc septum
Lat Humeral epicondyle
Ulnar n
Triceps m
Olecranon
Right Upper Extremity, Cross Section through Elbow Joint

Figure 1D: Right upper extremity cross section through elbow joint. At the elbow the ulnar nerve lies in the cubital tunnel surrounded by fascia. The median antebrachial cutaneous nerve branches are in the subcutaneous tissue adjacent to the branches of the basilic vein. This is a difficult and risky area in which to look for the ulnar nerve.

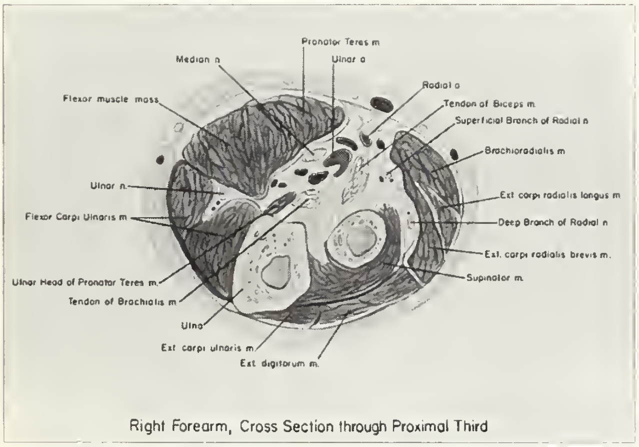

Pronator Teres m
Median n
Ulnar a
Flexor muscle mass
Radial a
Tendon of Biceps m
Superficial Branch of Radial n
Brachioradialis m
Ulnar n
Ext carpi radialis longus m
Flexor Carpi Ulnaris m
Deep Branch of Radial n
Ext. carpi radialis brevis m.
Ulnar Head of Pronator Teres m.
Supinator m.
Tendon of Brachialis m
Ulna
Ext carpi ulnaris m.
Ext digitorum m.
Right Forearm, Cross Section through Proximal Third

Figure 1E: Right forearm cross section through proximal third. Distal to the elbow the ulnar nerve lies between the two heads of the flexor carpi ulnaris muscle. The median nerve and radial and ulnar artery are beneath the remainder of the flexor muscle mass. The median nerve and vessels should always be identified prior to detaching the flexor muscle to prevent their injury.

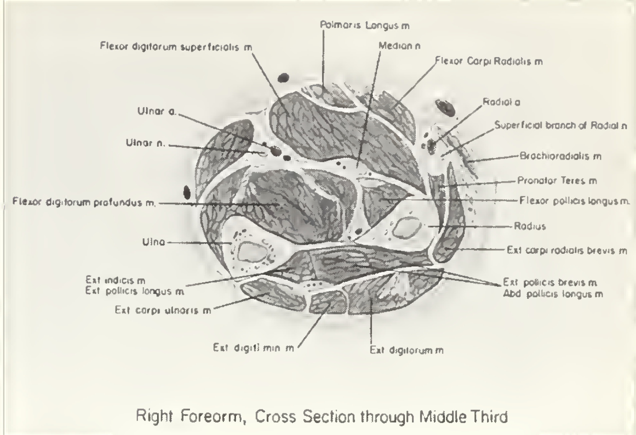

Palmaris Longus m
Flexor digitorum superficialis m
Median n
Flexor Carpi Radialis m
Radial a
Ulnar a.
Superficial branch of Radial n
Ulnar n.
Brachioradialis m
Pronator Teres m
Flexor digitorum profundus m.
Flexor pollicis longus m.
Radius
Ulna
Ext carpi radialis brevis m
Ext indicis m
Ext pollicis longus m
Ext pollicis brevis m
Abd pollicis longus m
Ext carpi ulnaris m
Ext digiti min m
Ext digitorum m
Right Forearm, Cross Section through Middle Third

Figure 1F: Right forearm cross section through middle third. In the mid forearm the ulnar nerve is joined by the ulnar artery as they pass to the wrist deep to the flexor carpi ulnaris and flexor digitorum superficialis muscles.

trauma such as a fracture or dislocation of the elbow. Most of the time conservative treatment provides only temporary relief because people are usually unable or unwilling to change jobs to avoid the activity producing the compression. Nocturnal splinting with the elbow in 15-20° flexion may be helpful in patients with mild symptoms. Constant paresthesia and/or muscle atrophy precludes the use of splints. Steroid injections probably have no meaningful role in the treatment of ulnar nerve compression at the elbow.

Surgical Treatment

Surgical treatments for ulnar nerve compression at the elbow have included decompression with or without subcutaneous or submuscular transposition and epicondylectomy. Each of these surgical techniques has its proponents, and there is no definitive randomized prospective study demonstrating the superiority of one technique. The American Society of Surgery of the Hand Committee on Nerve Compression currently recommends decompression and submuscular transposition as the treatment of choice. This is our preferred technique and will be discussed in detail below.

Simple decompression[9,10] has the advantage of being a simple and quick operation, whose disadvantages include lack of removal of the nerve from the site of compression and propensity for reentrapment of the nerve by scarring. Additionally, the nerve can sublux over the epicondyle, creating additional symptoms. This technique is only effective if the point of compression is within or adjacent to the cubital tunnel. Sites of compression higher in the arm or distal to the elbow would be inadequately treated. For these reasons the operation is rarely indicated.

Decompression and subcutaneous transposition removes the nerve from the cubital tunnel and results in decompression from the proximal arm to the belly of the flexor carpi ulnaris. The major disadvantage of anterior subcutaneous transposition is the superficial position of the nerve and its subsequent vulnerability to minor external pressure. This operation is also rarely indicated. Epicondylectomy[11,12,13,14] can adequately decompress the nerve in the area of the cubital tunnel. This operation has been widely practiced with reported good results.

Decompression with anterior submuscular transposition[15] allows decompression of the nerve throughout the entire course of potential impingement. Another advantage is relocation of the nerve into a well-padded and well-vascularized area. Transposition anterior to the elbow places the nerve under less tension where it should better tolerate repetitive flexion-extension movements. This operation eliminates the possibility of subluxation over the epicondyle and minimizes the risk of entrapment due to secondary scarring and is our procedure of choice.

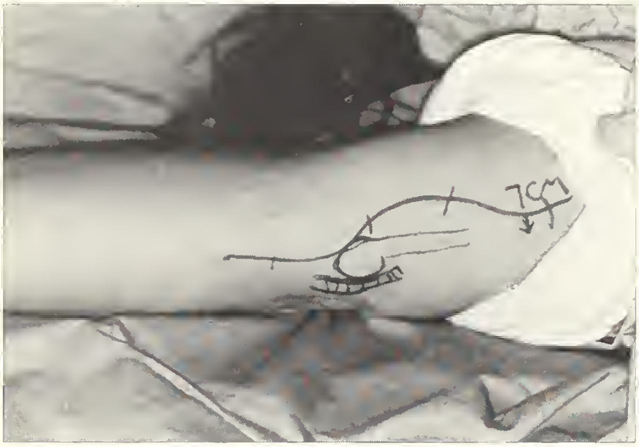


Figure 2A: Incision for decompression and submuscular transposition of the ulnar nerve at the elbow. The incision should be curved and should allow access to the ulnar nerve from 7 cm proximal to the medial epicondyle to approximately 6 cm distal to the cubital tunnel.

Surgical Decompression with Anterior Submuscular Transposition

The procedure can be carried out under general or axillary block anesthesia with tourniquet control. A curvilinear incision is performed on the medial aspect of the arm extending from 7-8 cm proximal to the medial epicondyle to 4 to 5 cm distal (Figure 2A). Skin flaps are developed anteriorly and posteriorly, exposing the basilic vein and its branches as well a the branches of the medial brachial and medial antebrachial cutaneous nerves (Figure 2B, C, D). The nerves should be meticulously preserved, as they can be a source of postoperative neuromas or hypesthesia (Figure 2E). The incision should be extended to expose the muscular fascia, particularly looking for the intermuscular septum proximally with the ulnar nerve lying posteriorly and the lacertus fibrosus distally in the forearm.

Decompression should be carried out from a proximal to distal fashion. The ulnar nerve is first identified in the distal arm just posterior to the intermuscular septum. The overlying fascia is incised, thereby decompressing the nerve in the distal arm. Care should be exercised to split the fascia proximally so as not to cause a new point of compression in the upper arm. As much of the mesoneurium as possible should be left attached to insure adequate perfusion of the nerve.

Tracing the nerve distally, one will notice its entrance into the cubital tunnel. Careful incising of the fibers bridging the medial epicondyle and the olecranon process should be performed, thereby freeing the nerve from the cubital tunnel (Figure 3A).

The nerve next passes between the humeral and ulnar heads of the flexor carpi ulnaris. The first branch to the flexor carpi ulnaris separates from the ulnar nerve

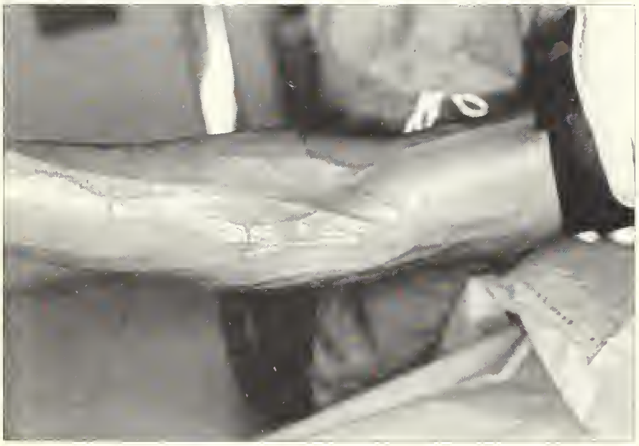


Figure 2B: Distribution of the basilic vein at the elbow and forearm. Compressing the tourniquet distends the veins and helps in designing the incision.

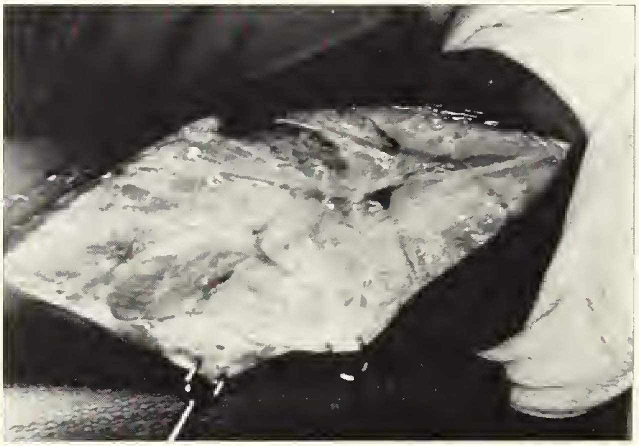


Figure 2C: Basilic vein and branches exposed at the elbow. These branches are helpful in identifying and protecting the branches of the median antebrachial nerve.

just distal to the cubital tunnel. Care should be taken to preserve its takeoff during dissection. The fascia and the muscle of the flexor carpi ulnaris should be carefully split in the direction of the fibers to allow mobilization of the nerve distally (Figure 3B).

Submuscular transposition begins with detaching the flexor pronator muscles from the medial epicondyle, leaving a cuff of fascia attached to the epicondyle to facilitate reapproximation once transposition is complete. One should be constantly aware of the close proximity of the median nerve at this point in the dissection. The ulnar nerve is next gently transposed into the subpronator position. With transposition great care should be taken to assure that there are no areas of kinking or acute angulation of the nerve (Figure 3C). Frequently the proximal muscular branches to the flexor carpi ulnaris will tether transposition of the nerve. These fascicles can usually be dissected for a distance from the main nerve trunk to allow a tensionless transposition (Figure 3D). The muscles are then reattached to the fascial strip left on the medial epicondyle (Figure 3E) A portion of the intermuscular septum is excised proximally in the arm to avoid compression at the point where the nerve passes from the anterior to the posterior compartment.

The tourniquet is released and hemostasis assured. The wound is closed with a single layer of running or interrupted nonabsorbable suture.

A bulky dressing is applied with an incorporated posterior splint maintaining the elbow at 90 degrees of flexion and the wrist in the neutral position. The first dressing change is at seven days then sutures are removed and the dressing is replaced with a removable plastic splint, which is worn for an additional five weeks. Three weeks postoperatively the patient begins passive extension exercises of the elbow four times per day. At six weeks the splint is discarded and active exercises of the elbow are begun. Patients are usually back to work in two to three months, depending on the strenuousness of their job.

The results of ulnar nerve decompression at the elbow are influenced not only by the technique but also by the extent of injury incurred by the nerve prior to decompression. Typically, numbness, tingling or paresthesia are relieved or significantly diminished. Intrinsic muscle atrophy usually persists except in the young.

Complications include hematomas, a painful scar or neuromas of the medial brachial or medial antebrachial cutaneous nerves. When failure to relieve symptoms occurs, extensive damage to the nerve may have occurred from the constriction at the entrapment site or the possibility of associated neuropathy should be entertained (e.g., alcoholic, diabetic).[16]

Ulnar Nerve Entrapment at the Wrist

Etiology

Causes of ulnar nerve entrapment at the wrist include direct and occupational trauma, tumors, vascular lesions, anomalous muscles and arthritis.[17,18] Single injuries can cause ulnar nerve compression at the wrist; however, repetitive trauma is more typical. The hypothenar hammer syndrome with ulnar artery an nerve symptoms usually results from use of the hand in a repetitive hammering motion such as pounding a hubcap on a wheel. This usually results in thrombosis of the ulnar artery if there is intimal disruption of an aneurysmal dilatation if medical disruption is the major component of injury. Ulnar nerve entrapment is often associated with the vascular injury. Any occupation

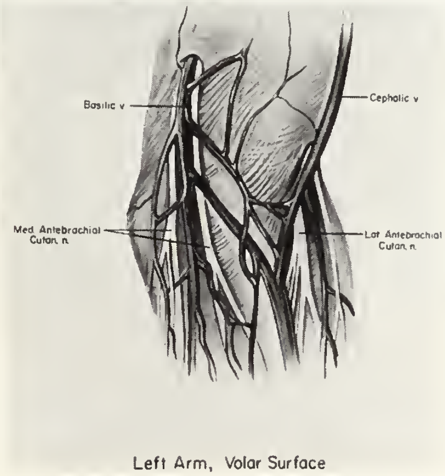


Figure 2D: The relationship of the median antebrachial cutaneous nerve to the branches of the basilic vein.

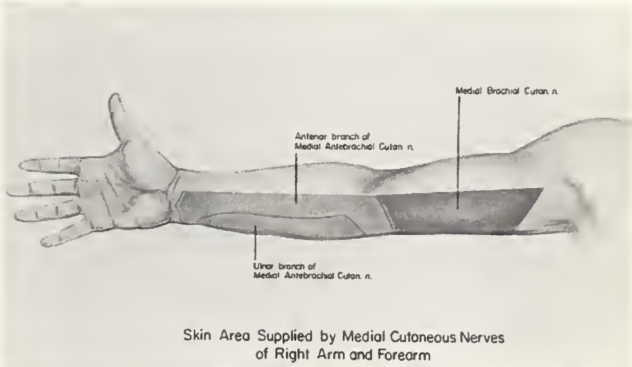

Medial Brachial Cutan. n.
Anterior branch of Medial Antebrachial Cutan. n.
Ulnar branch of Medial Antebrachial Cutan. n.
Skin Area Supplied by Medial Cutaneous Nerves of Right Arm and Forearm

Figure 2E: The distribution of the median antebrachial cutaneous nerve's two main branches. Injury to these branches produce hypesthesia or dysesthesia in the areas outlined. Painful neuromas are also common after injury to these nerves.

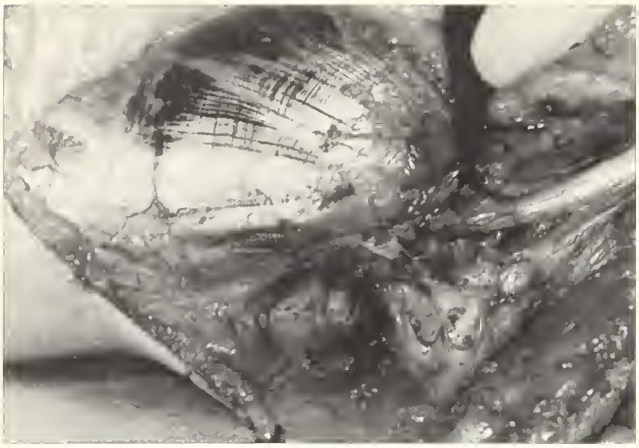


Figure 3A: Unroofing the cubital tunnel. The fascial layers overlying the ulnar nerve are divided from proximal to distal, keeping the nerve in view at all times.

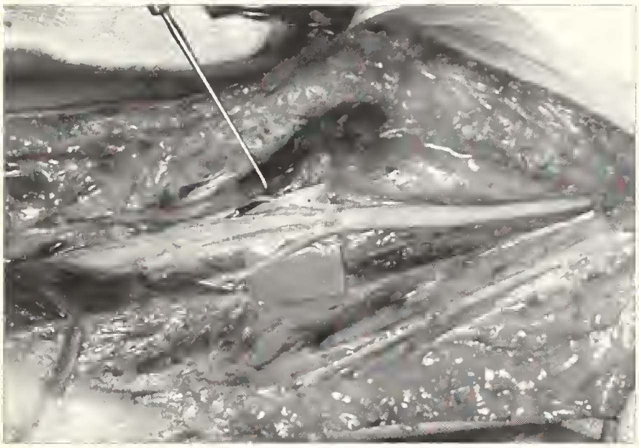


Figure 3B: The fascia overlying the flexor carpi ulnaris is split parallel to the muscle fibers and the ulnar nerve. Separating the muscle fibers exposes the nerve distal to the elbow. This should be done gently while looking for motor branches to the flexor carpi ulnaris.

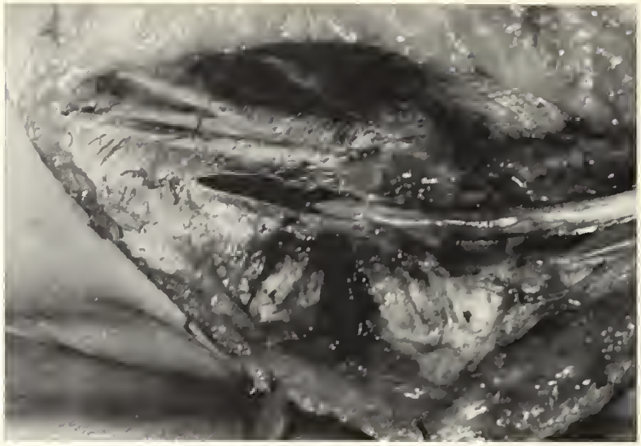


Figure 3C: After exposure of the intramuscular portion of the nerve is completed, the nerve can be mobilized from the cubital tunnel. Detaching the flexor muscle mass from the medial epicondyle now allows anterior and submuscular transposition of the nerve.

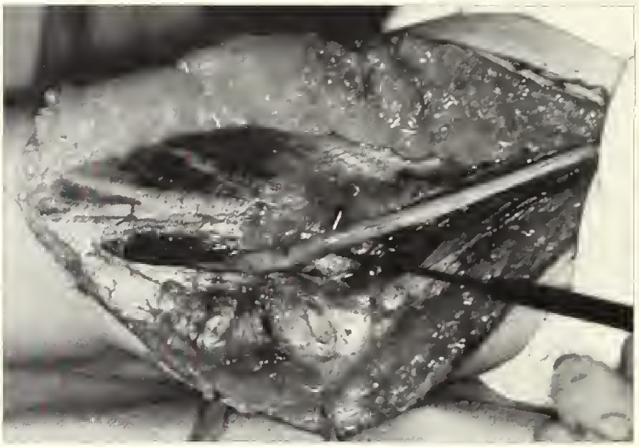


Figure 3D: Short motor branches to the flexor carpi ulnaris occasionally tether the nerve and prevent proper transposition or cause the nerve to be kinked. These branches should be dissected free from the main nerve trunk using magnification. They can be separated without damage to neural tissue for several centimeters. This added length will allow complete transposition without angulation.

which exposes the hypothenar area to repetitive injuries, vibration or pressure can also cause entrapment of the ulnar nerve in Guyon's canal.[19] Dislocations of the carpometacarpal joints,[20] the carpal joints or the radiocarpal joint can also produce ulnar nerve entrapment. Fractures of the hamate, triquetrum and base of the ring and little finger metacarpals are also associated with ulnar nerve symptoms. Ganglions[21,22,23,24] and lipomas[25] are the common tumors associated with ulnar nerve compression at the wrist. Vascular lesions include hemangiomas, vascular malformations, or arteriovenous fistulas. Anomalous muscles[26,27,28] which have been described as causes of ulnar nerve compression at the wrist include reverse palmaris longus, palmaris brevis profundus, and accessory palmaris longus, as well as anomalous origins of the flexor digiti minimi, abductor digiti minimi, and flexor carpi ulnaris.

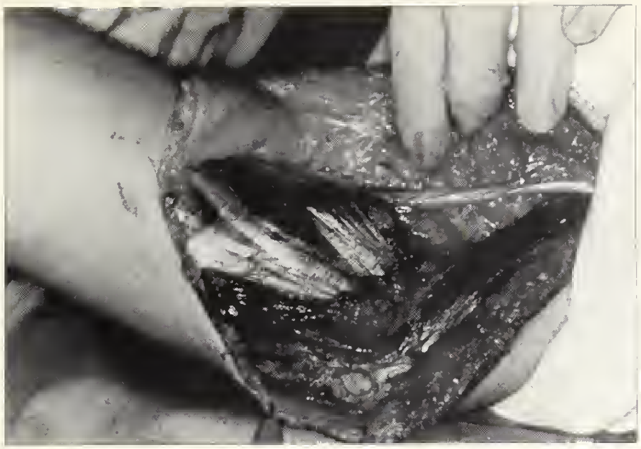


Figure 3E: After anterior transposition of the nerve, the elbow is flexed and the flexor muscle mass reattached to the medial epicondyle with nonabsorbable suture. The nerve is inspected proximally to be sure it is not kinked and the intermuscular septum resected. The tourniquet is deflated and hemostasis obtained prior to wound closure. The elbow is dressed in 90º of flexion with bulky dressing, which may incorporate a posterior splint. Drains are not used.

Pertinent Anatomy[29,30]

Distal to the elbow the ulnar nerve passes through the forearm in the space bounded ulnarly and volarly by the flexor carpi ulnaris, radially and volarly by the flexor digitorum superficialis, and dorsally by the flexor digitorum profundus (Figure 1D). The ulnar nerve is accompanied by the ulnar artery, with maintains it radial orientation throughout its course, and usually by a pair of vena comitantes flanking the artery. As the nerve approaches the radiocarpal joint, it lies volar to the pronator quadratus muscle and just dorsal to the tendon of the flexor carpi ulnaris muscle (Figure 4A).

Approximately 9 cm proximal to the wrist crease, the ulnar nerve gives off a major dorsal sensory branch, which winds ulnarly and dorsally to provide sensation to the dorsal aspect of the ring and little fingers. Just proximal to the wrist crease, the palmar sensory branch of the ulnar nerve pierces the superficial palmar fascia to provide sensation to the are over the hypothenar eminence.

The ulnar nerve displays an interesting fascicular arrangement as it courses from the forearm into the hand. Proximally there are three components: ulnarly a group of fascicles that branch off to form the dorsal sensory branch, a middle group of fascicles that will supply the deep motor branch, and a radial group that will supply the sensory branches to the little and ring fingers. After the separation of the dorsal sensory branch, the arrangement of the fascicles changes. Within Guyon's canal the now ulnar motor fibers spiral volarly to reach the radial aspect of the sensory fascicles and ultimately penetrate the fibrous arch of the flexor digitorum brevis muscle to innervate the intrinsic hand muscles.

The anatomy of Guyon's canal (Figure 4B) has been well described by Denman.[30] The canal begins at the proximal edge of the palmar carpal ligament and extends to the fibrous arch of the hypothenar muscles, totalling 4 to 4.5 cm in length. The dorsal surface of the canal is formed by the transverse carpal ligament. Ulnarly is the flexor carpi ulnaris tendon, the pisiform and the abductor digiti minimi muscle. Radially are the flexor tendons an the hook of the hamate. Volarly the structures contained in the canal lie vulnerable to any localized trauma. Distally in the canal they are covered only by the thin palmaris brevis muscle fibers, the superficial palmar fascia, the subcutaneous fat and the overlying skin (Figure 4C).

Diagnosis

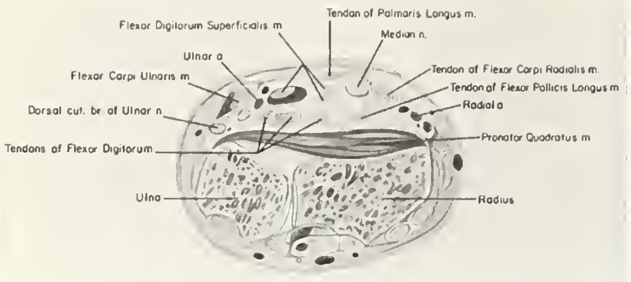
The diagnosis of ulnar nerve entrapment at the wrist can usually be established by a thorough history physical examination, and electrodiagnostic studies. Ancillary studies including x-rays and blood tests can be helpful in atypical case.

A thorough history should be obtained particularly attentive to the presence of numbness or tingling in the ring and little fingers. Cold intolerance and associated pallor or cyanosis in the ring and little fingers may also be present. The history should elicit the details of any preexisting trauma or arthritis and a clear documentation of the onset of the problem.

Physical examination often demonstrates subjectively-altered sensation per light tough or monofilament testing in the ring and little finger. If sensation is normal on the dorsum of the ring and little fingers, the site of compression is distal to the takeoff of the dorsal sensory branch. If sensation is abnormal on the dorsum of the ring and little fingers, the site of compression is usually at the elbow. Advanced cases of compression will often have associated atrophy of the hypothenar and intrinsic muscles. An Allen test should always be performed as part of the evaluation of ulnar nerve symptoms at the wrist. This will detect most cases of ulnar artery thrombosis (Figure 5A,B). A Tinel's sign may be present over the site of compression.

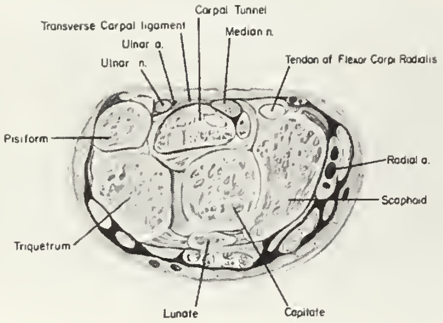
If there is a history of trauma, x-rays of the wrist including a carpal tunnel view should be obtained to exclude a fracture or dislocation. Fractures of the hook of the hamate can be particularly hard to exclude. If routine x-rays or a carpal tunnel view do not reveal a fracture an there is significant tenderness in the area of the hook of the hamate, a bone scan or CT scan should be obtained to exclude the diagnosis.

Electrodiagnostic studies[6] will usually localize the


Tendon of Palmaris Longus m.
Flexor Digitorum Superficialis m
Median n.
Ulnar a
Flexor Carpi Ulnaris m
Tendon of Flexor Carpi Radialis m.
Tendon of Flexor Pollicis Longus m
Radial a
Dorsal cut. br. of Ulnar n
Pronator Quadratus m
Tendons of Flexor Digitorum
Ulna
Radius

Right Forearm, Cross Section through Distal Third

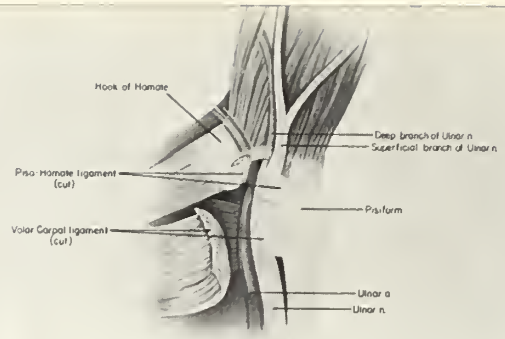
Figure 4A: Approximately 9 cm proximal to the flexion crease of the wrist, the dorsal sensory branch separates from the ulnar nerve and passes beneath the flexor carpi ulnaris tendon to provide sensation to the dorsum of the ring and little fingers. At this level the motor fibers are ulnar to the palmar sensory fibers.


Carpal Tunnel
Transverse Carpal ligament
Median n.
Ulnar a.
Ulnar n.
Tendon of Flexor Carpi Radialis
Pisiform
Radial a.
Scaphoid
Triquetrum
Lunate
Capitate

Right Hand, Cross Section through First Carpal Row

Figure 4B: The palmar root of Guyon's canal is formed proximally by the volar carpal ligament and the pisohamate ligament distally. The ulnar artery is radial to the nerve. Within the tunnel the motor fibers spiral around the sensory fibers in a volar and radial direction, passing deep to innervate the intrinsic muscles. The now ulnarmost palmar sensory fibers continue distally in the subcutaneous tissue to innervate the ulnar half of the ring and both sides of the little finger. Within the canal the nerve can be compressed at any point from the entrance to the fibrous arch of the hypothenar muscles.

point of compression to the wrist. Arteriography is not necessary unless there is suspicion of a vascular lesion. If ulnar artery thrombosis or aneurysm is present and reconstruction is anticipated due to distal insufficiency,


Hook of Hamate
Deep branch of Ulnar n
Superficial branch of Ulnar n
Piso-Hamate ligament (cut)
Pisiform
Volar Carpal ligament (cut)
Ulnar a
Ulnar n.

Guyon's Canal (Left Hand)

Figure 4C: Cross section of wrist demonstrating the boundaries of Guyon's canal and the relationship of the ulnar nerve and artery.

an arteriogram can be helpful to assess the distal vasculature. Otherwise, ligation and excision is the treatment of choice.

Conservative Treatment

Conservative treatment for ulnar nerve entrapment at the wrist is usually unsuccessful. The reason for failure is largely due to the etiology of the compression. Refraining from the activity producing compression may produce improvement in symptoms, particularly in neuropathy associated with bicycle riding.

Surgical Treatment

Surgical treatment usually falls into one of three categories. The first includes those patients with sensory and/or motor symptoms related to ulnar nerve entrapment at the wrist. Thee cases are treated by surgical decompression of the canal of Guyon from the distal forearm through the canal including the fibrous arch of the hypothenar muscles to insure decompression of the motor branch. The second category includes those patients with associated vascular problems. In addition to ulnar nerve compression, these patients usually require either resection of a thrombosed ulnar artery,[31] aneurysm[32,33] or vascular malformation. These vascular problems may require simply resection and ligation of the ulnar artery or bypass grafting, depending on the adequacy of circulation to the hand. The third category of patients includes those with ulnar nerve symptoms associated with carpal tunnel syndrome.[34] Most of these patients are adequately treated by decompression of the carpal tunnel alone. Once the carpal tunnel is decompressed by transection of the transverse carpal ligament, the oval-shaped Guyon's canal takes on a more circular

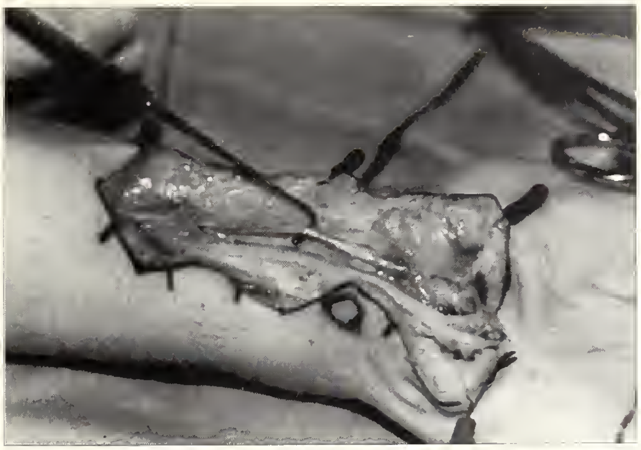


Figure 5A: Thrombosed ulnar artery associated with ulnar nerve compression at the wrist.

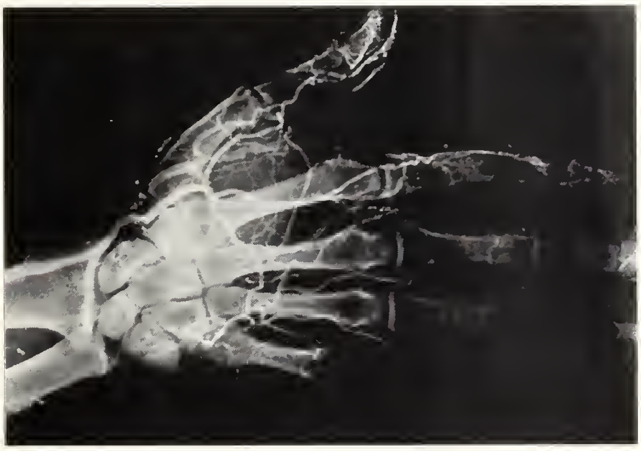


Figure 5B: Arteriogram of the patient shown in Figure 5A demonstrating occlusion of the ulnar artery and the digital arteries in the long, ring and little fingers. Note that the ulnar artery is thrombosed proximally to the first patent vessel in the forearm.

shape, thereby effecting a release of tension across the canal.[35] If there is a Tinel's sign over the ulnar nerve and slowed conduction across the canal of Guyon or intrinsic wasting in the ulnarly-innervated muscles, the ulnar nerve should be decompressed through the same incision used for decompression of the median nerve.

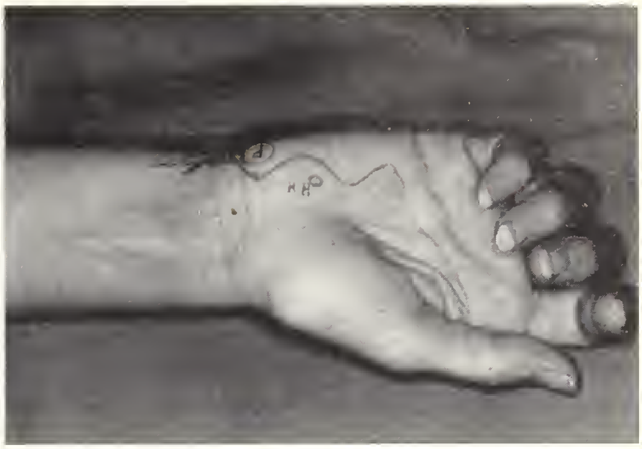


Figure 6A: Incision for decompression of the ulnar nerve at the wrist. Note the relationship of the incision to palpable landmarks including the flexor carpi ulnaris tendon, pisiform and hook or hamate.

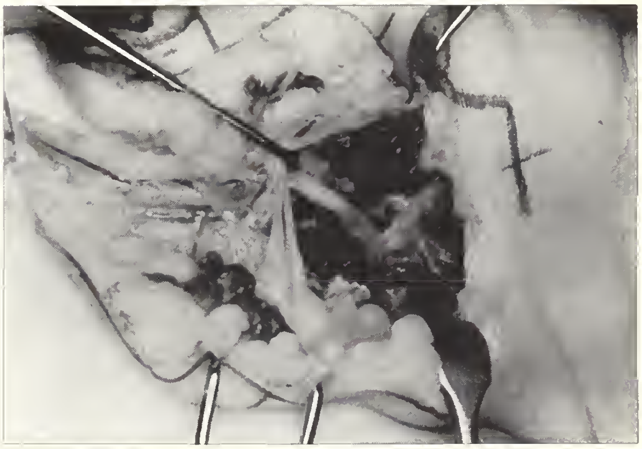


Figure 6B: This figure demonstrates the bifurcation of the ulnar nerve into sensory and motor branches. The sensory branches are passing into the palm. The motor branches ar passing deep beneath to innervate the intrinsic hand muscles. The fibrous arch of the hypothenar muscles has been divided.

Surgical Decompression at the Wrist

The incision is begun 2 to 3 cm proximal to the volar wrist crease. A line is drawn from this point along the radial border of the flexor carpi ulnaris tendon toward the ring finger onto the hypothenar area for a distance of approximately 2 cm (Figure 6A). The incision should have a zig-zag configuration so as to minimize postoperative contractures. The ulnar nerve should be found proximally lying under the flexor carpi ulnaris tendon. Division of the volar carpal ligament and the pisohamate ligament exposes the bifurcation of the ulnar nerve into its deep motor branch and the sensory branches to the ring and little fingers. The motor branch then passes between the abductor digiti minimi and flexor digiti minimi and pierces the opponens digiti minimi. The superficial branches continue in a subcutaneous plane into the little and ring fingers (Figure 6B).

Decompression of the ulnar nerve can be performed at the same time as releases of the carpal tunnel for median nerve compression. Care must be taken to not transect the palmar cutaneous branch of the ulnar nerve with the ulnar extension of the dissection that is necessary.

Postoperatively the patient is placed in a volar splint for three weeks with the wrist in neutral position, leaving the fingers and thumb free. Subsequently the splint is discarded with gradual increase in activity.

Complications of release of Guyon's canal are rare. A persistently-tender scar is the most common early complication. This usually resolves in three to six months. neuromas of the palmar cutaneous branch of the ulnar nerve have been described. Failure of release of the canal into the distal forearm can result in continued entrapment postoperatively. ■

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St. Louis, Missouri 63110

■ **Claude R. Bruner, M.D.,** Camdenton, Missouri, died February 2, 1992 at age 92. Born in north Missouri, Dr. Bruner attended medical schools at the University of Missouri and Northwest University receiving his medical degree 1927. Dr. Bruner, an opthomologist, practiced in Columbia more than 35 years, serving on the faculty of the University of Missouri Medical School and on the staff of both University Hospital and Clinics and Boone Hospital Center. He was an honor member of Boone County Medical Society and a member of the American Academy of Ophthalmology and Otolaryngology.

■ **Max Deutch, M.D.,** St. Louis, Missouri, died January 1, 1992 at age 86. Born in St. Louis, Dr. Deutch attended Washingtion University and received his medical degree from Washington University Medical School in 1926. He was named associate professor of clinical pediatrics at Washington University School of Medicine in 1937, and honored last spring by St. Louis Children's Hospital for his 50 years of service. After retirement from private practice, he continued to practice pediatrics working at the city's well-baby clinics in several St. Louis County school districts, the Missouri School for the Blind and at both the city and county health departments. Dr. Deutch was a member of the Delta Gamma Foundation for the Visually Handicapped Children and was on the board of directors for Talking Tapes for the Blind, and a member of the American Academy of Pediatrics.

■ **Penco T. Dzundzev, M.D.,** St. Louis, Missouri, died January 17, 1992 at age 66. Dr. Dzundzev received his medical degree from Zagrab University in Yugoslavia in 1955. He arrived in St. Louis in 1960, where he became a resident at Lutheran Hospital. He also worked at the Truman Restorative Center before opening a his as a practice as a family physician in St. Louis Hills. Dr. Dzundzev's latest practice was in the Hampton Village Medical Building. He was a member of Missouri State Medical Association and American Geriatrics Society, Inc.

■ **William C. Hummel, M.D.,** St. Louis, Missouri, died January 1, 1992 at age 56. Dr. Hummel attended the University of Notre Dame and received his medical degree from St. Louis University School of Medicine in 1961. He had been on staff of Berland Radiology Associates, Ltd., since 1965. Dr. Hummel was a former president of Greater St. Louis Society of Radiologists, a member of the American Medical Association, Radiological Society of North America, Missouri Radiological Society, and the American College of Radiology.

■ **George M. Klingner, M.D.,** Turners, Missouri, died February 6, 1992 at age 80. Born in Springfield, Missouri, Dr. Klingner, attended Drury College and received his medical degree from Washington University School of Medicine-St. Louis in 1936. Dr. Klingner practiced medicine in Springfield for 50 years. He was on the medical staff of Cox Medical Center and St. John's Hospital. He was a member of the Greene County Medical Society, Missouri State Medical Association, Southern Medical Association, and the American Academy of Family Physicians.

■ **Robert S. Weinhaus, M.D.,** St. Louis, Missouri, died December 31, 1991 at age 69. Dr. Weinhaus received his medical degree from Washington University School of Medicine in 1945. He practiced internal medicine until a back injury curtailed his work. Dr. Weinhaus then became a psychiatrist and taught psychiatry at St. Louis University. He was a member of American College of Physicians, American Geriatrics Society, Inc., American Pyschosomatic Society, and American Physicians Fellowship, Inc.

■ **Daniel L. Yancey, M.D.,** Springfield, Missouri, died February 6, 1992 at age 88. Born in Saline County, Missouri, Dr. Yancey attended Missouri Valley College and received his medical degree from St. Louis University School of Medicine in 1930. Dr. Yancey was Springfield's first board-certified orthopedic surgeon. His association with the Smith-Glynn-Callaway Clinic lasted 45 years. Dr. Yancey helped establish a crippled children's program at Burge Hospital, now Cox Medical Center, and served as director of the State Crippled Children's Service. He was a life member of the American College of Surgeons, the American Academy of Orthopaedic Surgeons, the Association of Military Surgeons, National Rehabilitation Association, Clinical Orthopaedic Society and the Southwest Surgical Congress. He was a founding member of the Orthopaedic Research and Education Foundation, and a former president of Mid-Central States Orthopaedic Society, the St. Louis Orthopaedic Society and Greene County Medical Society.

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Doctor's Name

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Office Phone (To be used only if added info is needed)
(   )

Current Medical Specialty(ies)

Former Medical Specialty(ies) (for past 10 years)

List surgical procedures routinely performed

State(s) where currently licensed

States where formerly licensed (for past 10 years)

Amount of Professional Liability Insurance now carried
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02

What is a residency?

by
Hugh E. Stephenson Jr., M.D.

Technically speaking, formal medical education beyond the MD degree is usually classified as residency education. Substantial clinical experience is provided the "resident" as he or she prepares to become competent in one of the various recognized medical or surgical specialties. Completion of the formal training program usually insures eligibility to take the certifying examination offered by that particular program. Many state licensing bodies and certifying agencies rely on accreditation to assure the accepted standards of training have been met. Hospitals and other credentialling agencies look at the accreditation as an "indication of satisfactory professional education and competence".

Currently, there are 24 residency review committees, all sponsored by at least two or three organizations such as the American Medical Association's Council on Medical Education and the American Board of Dermatology.

The Council on Medical Education of Hospitals of the American Medical Association first published a list of hospitals approved for internships in 1914. Since then, post-graduate programs have been evaluated continuously. In 1974, five member organizations formed the Liaison Committee on Graduate Medical Education (LCGME) to achieve policies and coordination among the several residency review committees. This organization was renamed the Accreditation Council for Graduate Medical Education (ACGME) in 1981. The five member organization of the ACGME are the American Board of Medical Specialties, American Hospital Association, American Medical Association, Association of American Medical Colleges, and the Council on Medical Specialties Societies. Each organization has four voting representatives on the 24 - seat ACGME Council. In addition, the resident representative has a vote and a public representative has a vote on the council. Non-voting representatives from the federal government and from the Chair of the Residency Review Committee Council are present. Generally speaking, all decisions are made by the Residency Review Committees, except for contested decisions, which are referred to the ACGME as the final decision-making body.

Today there are over 6,700 residency programs in 24 major specialty areas and 49 other specialized training areas. Over 1,500 institutions sponsor these programs in which 90,000 residents each year are enrolled.

For a particular residency program to be approved, the residency review committee responds to an application for accreditation by the program and subsequently, the process includes a scheduled on-site review. The initial accreditation is re-evaluated in approximately two years and they are reviewed on a cycle which should not exceed five years.

Accreditation provides assurance of nationally accredited standards for program quality, recognition of their education by their professional peers and eligibility in most instances for state licensure and/or professional certification.

The essentials for an accredited residency and graduate medical education contain general requirements applicable for all residency education programs. There are 81 different specialty areas, for which special requirements are applicable.

The ACGNME develops the general requirements and the special requirements are generally developed by the residency review committees with consultation and approval by the ACGME.

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■ **Kerry K. Assil, M.D.,** 3655 Vista Ave., St. Louis,MO 63110, has become a member of the St. Louis Metropolitan Medical Society. Dr. Assil received his medical degree from the University of California at San Diego, CA, in 1986. He completed his residency at Bethesda Eye Institute, St. Louis University, St. Louis, MO. Dr. Assil specializes in ophthalmology.

■ **William L. Becker, M.D.,** 135 W. Adams, Kirkwood, MO 63122, has become a member of the St. Louis Metropolitan Medical Society. Dr. Becker received his medical degree from Washington Univesity, St. Louis, MO, in 1987. He completed his residency at Washington University, Barnes Hospital, St. Louis, MO. Dr. Becker specializes in ophthalmology.

■ **James H. Boyd, M.D.,** 3635 Vista Ave. at Grand, St. Louis, MO, has become a member of the St. Louis Metropolitan Medical Society. Dr. Boyd received his medical degree from the University of Missouri-Columbia, MO, in 1985. He completed his residency at the University of Missouri-Columbia Hospitals & Clinics, Columbia, MO. Dr. Boyd specializes in otolaryngology.

■ **James P. Crane, M.D.,** 660 S. Euclid, Box 8081, St. Louis, MO, has become a member of the St. Louis Metropolitan Medical Society. Dr. Crane received his medical degree from Indiana University, Indianapolis, IN, in 1970. He completed his residency at Barnes Hospital, St. Louis, MO. Dr. Crane specializes in obstetrics and gynecology.

■ **Margarethe, Hagemann (Williams), M.D.,** 4444 Forest Park, P.O. Box 66828, St. Louis, MO 63166, has become a member of the St. Louis Metropolitan Medical Society. Dr. Hagemann received her medical degree from Medical College of Wisconsin, Milwaukee, WI, in 1971. She completed her residency at the Medical College of Wisconsin. Dr. Hagemann specializes in internal medicine.

■ **Philip E. Higgs, M.D.,** One Barnes Hospital Plaza, Suite 17242, St. Louis, MO 63110, has become a member of the St. Louis Metropolitan Medical Society. Dr. Higgs received his medical degree from the University of Florida, Gainesville, FL, in 1974. He completed his residency at Washington University, Barnes Hospital, St. Louis, MO. Dr. Higgs specializes in plastic surgery.

■ **Manny S. Iyer, M.D.,** 5177 Broadway, Kansas City, MO 64111, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Iyer received his baccalaureate degree from Loyola College, Madras, India, and obtained his medical degree from Stanley Medical College, Madras, India, in 1978. He completed his residency at Truman Medical Center, Kansas City, MO. Dr. Iyer specializes in Plastic and Reconstructive Surgery.

■ **Garry A. Johnson, M.D.,** 450 N. New Ballas, Suite 103, Creve Couer, MO 63141, has become a member of the St. Louis Metropolitan Medical Society. Dr. Johnson received his medical degree from the University of California-Davis, CA, in 1980. He completed his residency at the Oregon Health Sciences Center, Portland, OR. Dr. Johnson specializes in anesthesiology.

■ **Gary D. Koenig, M.D.,** 11155 Dunn Rd., St. Louis, MO 63136, has become a member of the Missouri State Medical Association. Dr. Koenig received his baccalaureate degree from Vanderbilt University, Nashville, TN, and obtained his medical degree from St. Louis University, St. Louis, MO, in 1985. He completed his residency at St. Mary's Health Center, St. Louis, MO. Dr. Koenig specializes in internal medicine.

■ **Robert S. Kramer, M.D.,** 522 N. New Ballas, St. Louis, MO 63141, has become a member of the St. Louis Metropolitan Medical Society. Dr. Kramer received his medical degree at Washington University, St. Louis, MO, in 1983. He completed his residency at the University of Minnesota Health Sciences Center, Minneapolis, MN. Dr. Kramer specializes in orthopedic surgery.

■ **Anne N. Lass, M.D.,** 1465 S. Grand, St. Louis, MO 63104, has become a member of the St. Louis Metropolitan Medical Society. Dr. Lass received her medical degree from the Chicago Medical School, North Chicago, IL, in 1981. She completed her residency at the University of Chicago. Dr. Lass specializes in pediatrics.

■ **Rudy G. Madlang, M.D.,** P.O. Box 975, Columbia, MO 65205, has become a member of the Boone County Medical Society. Dr. Madlang received his baccalaureate degree from Northwestern University, Evanston, IL, and obtained his medical degree from The Chicago Medical School, Chicago, IL in 1979. He completed his residency at University of California-Los Angeles, CA. Dr. Madland specializes in anesthesiology.

NEW MEMBERS

Continued from page 245

■ **Robert F. Onder, M.D.,** 11722 Studt Ave., St. Louis, MO 63141, has become a member of the St. Louis Metropolitan Medical Society. Dr. Onder received his medical degree from Washington University, St. Louis, MO, in 1987. He completed his residency at Barnes Hospital, St. Louis, MO. Dr. Onder specializes in internal medicine.

■ **Chris M. Pinkham, M.D.,** 5400 N. Oak Trafficway, Suite 200, Kansas City, MO 64118, has become a member of the Clay-Platte County Medical Society. Dr. Pinkham received his baccalaureate degree from Wichita State University, Wichita, KS, and obtained his medical degree from the University of Kansas, Larwrence-Kansas City, KS, in 1988. He completed his residency at the University of Kansas Medical Center, Kansas City, KS. Dr. Pinkham specializes in family practice.

■ **Bernard C. Randolph, Jr., M.D.,** 1617 S. 3rd, St. Louis, MO 63104, has become a member of the Missouri State Medical Association. Dr. Randolph received his baccalaureate degree from Amherst College, Amherst, MA and obtained his medical degree from Howard University College of Medicine, Washington DC, in 1985. He completed his residency at Jewish Hospital of St. Louis, MO. Dr. Randolph specializes in physical medicine and rehabilitation.

■ **Brian G. Rubin, M.D.,** 216 South Kingshighway, St. Louis, MO 63110, has become a member of the St. Louis Metropolitan Medical Society. Dr. Rubin received his medical degree from the University of Vermont College of Medicine, Burlington, VT, in 1984. He completed his residency at Yale-New Haven Hospital, New Haven, CT. Dr. Rubin specializes in general surgery.

■ **Marvin P. Steiner, M.D.,** 17611 E. 24th Highway, Independence, MO 64056, has become a member of the Missouri State Medical Association. Dr. Steiner received his baccalaureate degree from the University of Illinois, Chicago, IL, and obtained his medical degree from the Chicago Medical School, North Chicago, IL, in 1975. He completed his residency at the Methodist Medical Center, Peoria, IL. Dr. Steiner specializes in family practice.

■ **James G. Stewart, M.D.,** 3450 Bridgeland Drive, Bridgeton, MO 63044, has become a member of the St. Louis Metropolitan Medical Society. Dr. Stewart received his medical degree from the Mayo Medical School, Rochester, NY, in 1984. He completed his residency at the University of Chicago, Chicago, IL. Dr. Stewart specializes in radiology.

■ **Eric J. Stuphen, M.D.,** 6150 Oakland Ave., St. Louis, MO 63139, has become a member of the St. Louis Metropolitan Medical Society. Dr. Stuphen received his medical degree from St. Louis University, St. Louis, MO, in 1987. He completed his residency at St. Mary's Hospital, San Francisco, CA. Dr. Stuphen specializes in radiation oncology.

■ **Cheng Chang Tsai, M.D.,** 6150 Oakland Ave., St. Louis, MO 63139, has become a member of the Missouri State Medical Association. Dr. Tsai received his medical degree from Kaohsiung Medical College, Daohsiung, Taiwan in 1968. He completed his residency at Washington University, Barnes Hospital, St. Louis, Missouri. Dr. Tsai specializes in pathology.

RESIDENTS: Michael N. Stiffman, M.D., *Family Practice* & Kent D. Walker, M.D., *Dermatology*

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Carcinogenesis, Mutagenesis, Impairment of Fertility–A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups.The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy–Teratogenic Effects–Pregnancy Category C–Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers–Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use–Safety and effectiveness in children have not been established.

Use in Elderly Patients–Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic–Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular–In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS–Rare cases of reversible mental confusion have been reported.

Endocrine–Clinical pharmacology studies and controlled clinical trials showed no evidence of anti-androgenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely

Hematologic–Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental–Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity–As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other–Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

PV 2093 AMP [101591]

Additional information available to the profession on request

Lilly

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YOCON®

YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

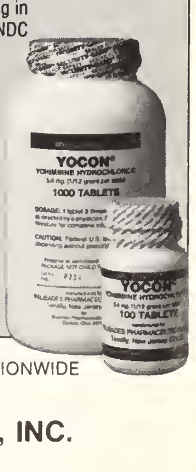
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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UNIVERSITY OF MISSOURI-COLUMBIA

Through a joint effort with Harry S. Truman Memorial Veteran's Hospital in Columbia, MU's Ellis Fischel Cancer Center has acquired $1.85 million in new equipment that will allow the institutions to offer advanced radiation surgery.

The cooperative agreement will make available to patients from the two hospitals and other Columbia area hospitals a new stereotaxic surgery system, a dual-beam linear accelerator, an X-ray simulator system and a record/verify system.

The stereotaxic surgery system treats cancers of the brain or abnormal vascular problems through radiation rather than surgery. The exact location of the radiation surgery is determined by a computerized program. The dual-beam linear accelerator destroys or weakens cancerous tumors with precise radiation streams while minimizing the destruction of healthy skin tissue. The X-ray simulator system produces a photographic X-ray that shows which part of the body will receive radiation based upon computerized data and allow therapists to create appliances that block radiation from areas of the body other than the treatment locations. The record/verify system verifies each individual radiation treatment is set up correctly and then records the treatment specifications.

William C. Allen, M.D., professor emeritus of family and community medicine, was named the 1991 Doctor of the Year by the Boone County Medical Society. His most known activity is directing the Preceptorship Program at MU. The program places third-year medical students with rural preceptors around the state who incorporate the students into their practices.

Judith A. Cole, Ph.D., a instructor in the department of pharmacology, and **Robert W. Lim, M.D.,** assistant professor of pharmacology, both have received a FIRST Award from the National Institutes of Health. Dr. Cole's award will go to her research on parathyroid hormones as mechanisms of action in kidneys. Dr. Lim's grant will be used for his research in the growth factor regulation of muscle differentiation. The FIRST Award from the NIH is awarded to promising researchers who have not previously received significant funding from the NIH.

Donald Doll, M.D., professor of hematology and oncology, was awarded the 1991 Golden Chalk Award by the Graduate Professional Council. The award is given by students to a member of each graduate and professional school for their outstanding contribution to student education.

Leonard R. Forte, Ph.D., professor of pharmacology, recently presented a lecture and chaired a symposium at the 10th World Congress on Animal, Plant and Microbial Toxins in Singapore. Dr. Forte also gave a lecture at the University of Western Australia.

David Goldstein, M.D., professor of pediatrics and director of the Division of Pediatric Endocrinology/Diabetes, has been chosen for inclusion in the upcoming publication, The Best Doctors in America.

Thomas Hurley, M.D., associate professor of child health and physiology, was appointed to the Oral Biology and Medicine Study Section of the NIH. Members of the study section review grant applications, make recommendations to the appropriate national advisory board, and survey the status of research in their fields of science.

Gerald Perkoff, M.D.

Gerald T. Perkoff, M.D., professor emeritus of family and community medicine, has been named the 1992 Curtis G. Hames Research Award recipient by the Society of Teachers of Family Medicine, honoring his career accomplishments and dedication to research in family medicine. The award committee specifically noted Perkoff's research into the muscle disease, hereditary interstitial nephritis and the effects of prepaid group practices on the delivery of health-care services. The Hames Award is supported by the Hames Endowment of the department of family medicine at the Medical College of Georgia.

Edith Mitchell, M.D., associate professor of medicine, and **Amolak Singh, M.D.,** associate professor of radiology, were recognized by Cytogen Corp. for their work with radioimmunoscintigraphy and the development of OncoScint, a genetically created antibody used in diagnostic imaging in patients with colorectal and ovarian cancer. Dr. Mitchell also recently was honored for her service to the Missouri Department of Health, where she serves as clinical vice chairman of the Minority Health Advisory Committee.

Richard Oliver, Ph.D., director of the MU School of Health Related Professions, has been elected to the nomination committee for the American Society of Allied Health Professions. Oliver also is serving as vice chairman of the Midwest Association of Allied Health Deans in Academic Health Centers.

John T. Turner, Ph.D., assistant professor of pharmacology spoke in March at the annual meeting of the International Association of Dental Research in Boston. He presented a paper on aspects of the regulation of salivary gland function.

James M. Weiss, M.D., professor emeritus and former chairman of psychiatry, recently was awarded life fellowship in the American College of Psychiatrists and American Public Health Association.

Kim Wise, Ph. D., professor of molecular microbiology and immunology, recently received a five-year grant from the National Institute of Allergy and Infectious Disease. Wise is investigating the possibility that mycoplasmas could play a role as co-factors to the human immunodeficiency virus in the development of AIDS.

Washington University

LipoMatrix, Inc., a start-up company, will begin commercial development of a new breast implant technology pioneered at Washington University.

The new breast implant technology is based on a patent jointly developed by **Judy M. Destouet, M.D., John O. Eichling, M.D., Louis A. Gilula, M.D.,** and **Barbara S. Monsees, M.D.,** all radiologists with the Mallinckrodt Institute of Radiology at Washington University, and **V. Leroy Young, M.D.,** of the Division of Plastic and Reconstructive Surgery at Washington University's School of Medicine.

The patent describes the use of vegetable triglycerides (such as peanut oil) as radiolucent filler materials for breast implants. Unlike silicone gel and/or saline currently used as filler materials for breast implants, triglycerides appear not to interfere with mammograms. Another potential benefit of this technology is that the tryglyceride filler appears to be biocompatible in the event of an accidental leakage or rupture of the implant. Use of triglycerides as filler could also allow engineering of the filler material to satisfy texture demands.

Typically the investigations necessary to confirm the safety and efficacy of an implantable device for human use may require at least three years.

STATE LAWMAKERS RECOGNIZE FAMILY PHYSICIANS FOR 20 YEARS OF SERVICE

Representative Ken Lagen, Ben Koon, M.D., and Dr. Koon's daughter, Mary Nell Koon.

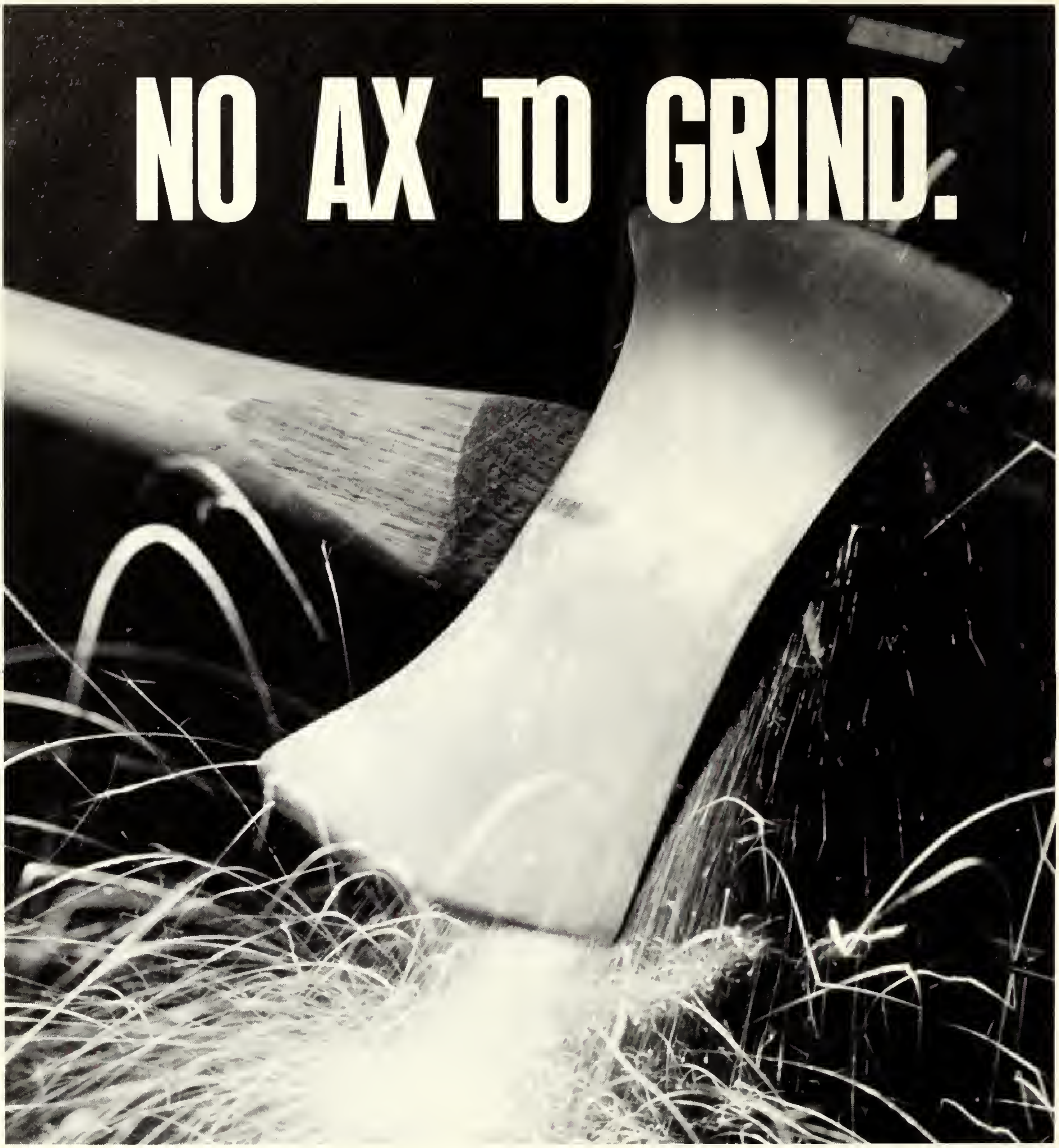
Senator Bob Johnson and Donald Potts, M.D.

1992 marks the twentieth year that the Missouri Academy of Physicians has coordinated the "Physician of the Day" program for Missouri's legislators.

On February 13th, Donald Potts, M.D., of Independence and Ben Koon, M.D., of Bolivar were presented with resolutions by Representatives W.T. Dawson and Kenneth Legan and Senators Bob Johnson and John Russell, which recognized the Missouri Academy for the 20-year tradition of sending a physician to the capitol to cover medical emergencies during the legislative session.

Assisted by Missouri State Medical Association's, Royal Cooper, the MAFP's "Physician of the Day" program began in 1972. Drs. Potts and Koon were the first family physicians to volunteer their services on the opening day of the 77th session and since that time Dr. Potts has served one or more days each year and Dr. Koon has also been a frequent participant.

Physician participants have gained insight into the legislative process and many legislators and staff members have benefited from their care.



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The continuing medical education accreditation program of the MSMA has full approval by the AMA's Council on Medical Education. The institutions and functions listed here qualify for Category I credit for the AMA Physician's Recognition Award. All accredited institutions in Missouri are invited to send this information for listing in *MISSOURI MEDICINE*. Please remember to include the name of the individual who can be contacted at your institution for additional information about your CME events.

APRIL

4 - *Chesterfield*: Colposcopy- LEEP- Laser. Contact Patty Fogertey, Program Coordinator, Department of Education, St. Luke's Hospital Institute for Health Education, 232 S. Woods Mill Rd., Chesterfield, MO, 63017, 314/542-4762.

4 - *St. Louis*: Liver Transplantation for the Primary Care Physician. Category 1 credit. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

7 to 9 - *St. Louis*: Doppler Echocardiography: The Quantitative Approach. 20 Category 1 credit hours. Contact Donna Reid, St. Louis University Medical Center, 314/577-8897.

13 - *St. Louis*: City-Wide Radiology Conference. Contact R. Gilbert Jost, M.D., Chief of Diagnostic Radiology, Mallindrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, MO 63110.

13 to 17 - *St. Louis*: Neuro CT Program. 25 Category 1 credit hours. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

15 & 16 - *St. Louis*: Advanced Laparoscopy for the General Surgeon. 15.75 Category 1 credit hours. Contact Washington University School of Medicine, Office of CME, Box 8063, 660 S. Euclid, St. Louis, MO 63110-1093, 314/362-6893.

17 - *Kansas City*: AIDS, The Second Decade. Contact The Ed Bixby Institute for Postgraduate Medical Education, Research Medical Center, 2316 E. Meyer Blvd., Kansas City, MO 64132, 816/276-4306.

20 & 24 - *St. Louis*: Advanced Cardiac Life Support Provider/Recert. 16 Category 1 credit hours. Contact David Wagner, St. Louis University Medical Center, 314/577-8521.

20 to 24 - *St. Louis*: The Medicolegal Death Investigator Training Course. 33 Category 1 credit hours. Contact Mary Fran Ernst, St. Louis University Medical Center, 314/522-6410 ext. 6509.

20 to 24 - *St. Louis*: Body CT Program. Category 1 credit. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

22 to 26 - *Washington D.C.*: Sixth Annual Critical Care Update. 35 credit hours in Category 1. For more information contact Svetlana Lisanti, Conference Administrator, Center for Bio-Medical Communication, Inc., 80 West Madison Avenue, Dumont, NJ, 07628. PH 201/385-8080.

24 to 26 - *St. Louis*: Practical Anatomy And Surgical Technique Workshop: Spinal Fracture Fixation. 20 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

24 & 25 - *St. Louis*: 5th Hearing Aid Conference. Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 South Euclid, St. Louis, MO 63110-1093. PH: 314/362-6893 or 800/325-9862.

25 - *St. Louis*: Third Annual Course - Management of Challenging Problems in Colon and Rectal Disease in 1992. 6 Category 1 credit hours. Contact Donna Troy, St. Louis University Medical Center, 314/577-8195.

24 to 26 - *Kansas City*: Workshop on Stress Echocardiography. Category 1 credit. Contact Beth M. Paul, Conference Coordinator, CME, St. Luke's Hospital. 816/932-2301.

27 to May 1 - *St. Louis*. Magnetic Resonance Imaging of Head & Body. Category 1 credit. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

27 to May 9 - *St. Louis*: Institute for Clinical Health Care Ethics. 78 Category 1 credit hours. Contact Donna Troy, St. Louis University Medical Center, 314/577-8195.

30 to May 1 - *Kansas City*: Current Management in Critical Care. Contact Linda F. Smutz, Kansas City Southwest Clinical Society, 7920 Ward Parkway, Suite 205, Kansas City, MO, 64114. PH 815/523-3383.

MAY

1 - *St. Louis*: Second William Harkins Lectureship. 4 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

2 - *St. Louis*: Third Annual Symposium on Hypercholesterolemia: Practical Aspects of Patient Management. 3 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

5 & 6 - *St. Louis*: Transesophageal Echocardiography Course. 13 Category 1 credit hours. Contact Dianne

Continued from page 253

Reid, St. Louis University Medical Center, 314/577-8897.

7 to 8 - *Kansas City*: Surgical Symposium - Advances in Surgical Care. Category 1 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

7 to 10 - *St. Petersburg, FL*:Emergency Department Legal Environment: Medicine's Response. 11 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

8 - *Kansas City*: Office Evaluation of Sleep Disorders. Category 1 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

11 - *St. Louis*: Interventional Radiology. A city-wide radiology conference by Daniel Picus, M.D. Contact R. Gilbert Jost, M.D, Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

12 - *St. Louis*: Pediatric Advanced Cardiac and Trauma Support & Pediatric Advanced Life Support Recert. Course. 7 Category 1 credit hours. Contact Mary Beth Foerstel, R.N., St. Louis University Medical Center, 314/577-5385.

13 - *St. Louis*: Update on Summer Sports and Sports Medicine. Contact Judith L. O'Guin, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141, 314/569-6290.

13 to 15 - *Kansas City*: Coronary Angioplasty for the Basic and Intermediate Dilator. Category 1 credit. Contact Beth M. Paul, Conference Coordinator, CME, St. Luke's Hospital. 816/932-2301.

14 & 15 - *Kansas City*: 24th Annual Matt Weis Symposium - Advances in Obstetrics & Gynecology. Contact Judith L. O'Guin, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141, 314/569-6290.

15 & 16 - *St. Louis*: Advanced Laparoscopy for the General Surgeon. Catagory 1 credit and endorsed by the SAGES. Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 South Euclid, St. Louis, MO 63110-1093. PH: 314/362-6893 or 800/325-9862.

15 to 17 - *St. Joesph*: Advanced Cardiac Life Support Provider Course. 22 Category 1 credit hours. Contatct Barb Tunks, R.N., Heartland Health System Critical Care Educator, 801 Faraon, St. Joesph, MO 64501, 816/271-7859.

15 to 19 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop: Advanced Microsurgery of the Lumbar & Cervical Spine. 36 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

18 to 22 - *St. Louis*: Body CT Program. Category 1 credit. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

18 to 23 - *St. Louis*: Health Care Ethics in a Pluralistic Society. 43 Category 1 credit hours. Contact Donna Troy, St. Louis University Medical Center, 314/577-8195.

27 to 29 - *Kansas City*: Annual Update in OB/GYN. Jointly sponsored with Truman Medical Center. 16 Category 1 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

JUNE

2 to 4 - *St. Louis*: Doppler Echocardiography: The Quantitative Approach. 20 Category 1 credit hours. Contact Dianne Reid, St. Louis University Medical Center, 314/577-8897.

3 - *St. Louis*: Pulmonary Embolus in Thromembolic Disease. Contact Judith L. O'Guin, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141, 314/569-6290.

4 to 6 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part I: Peripheral Nerve. 22 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

4 to 7 - *St. Louis*: Advances in Aesthetic & Reconstructive Breast Surgery. Contact Cathy Caurso, Office of CME, Washinton University School of Medicine, Box 8063, 660 S. Euclid, St. Louis, MO 63110-1093. PH 800/325-9862, 314/362-6893.

6 - *Kansas City*: Fourth Annual Lipid Conference. Jointly sponsored with UMKC School of Medicine. 3 Category 1 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

6 - *St. Louis*: New Concepts in the Management of Parkinson's Disease. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

7 to 9 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part II: Spine-Microsurgery & Stabilization. 24 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

8 to 12 - *St. Louis*: Neuro CT Program. 25 Category 1 credit hours. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

10 - *Kansas City*: Domestic Violence Awareness. 3 Category 1 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

10 to 14 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part III: Microsurgery of the Brain. 36 Category 1 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

CLASSIFIED ADVERTISEMENTS

Physicians Wanted

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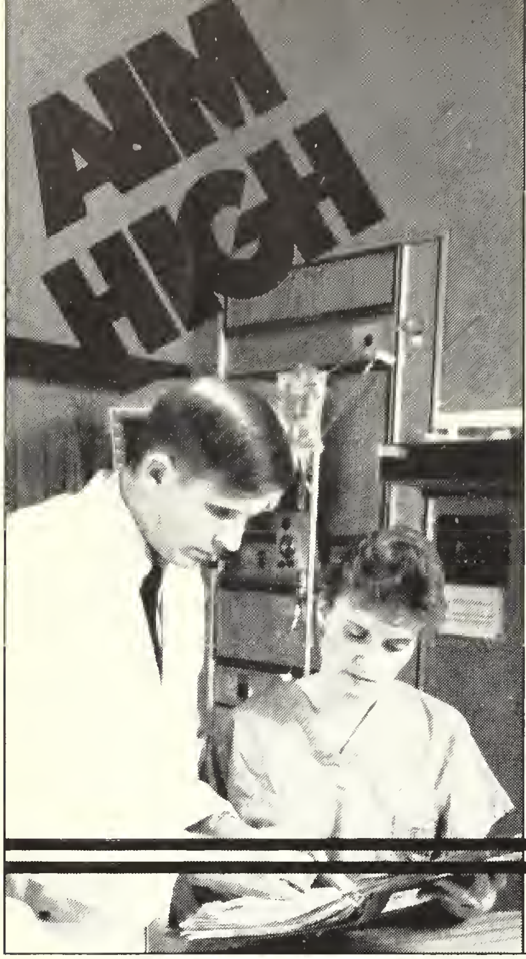

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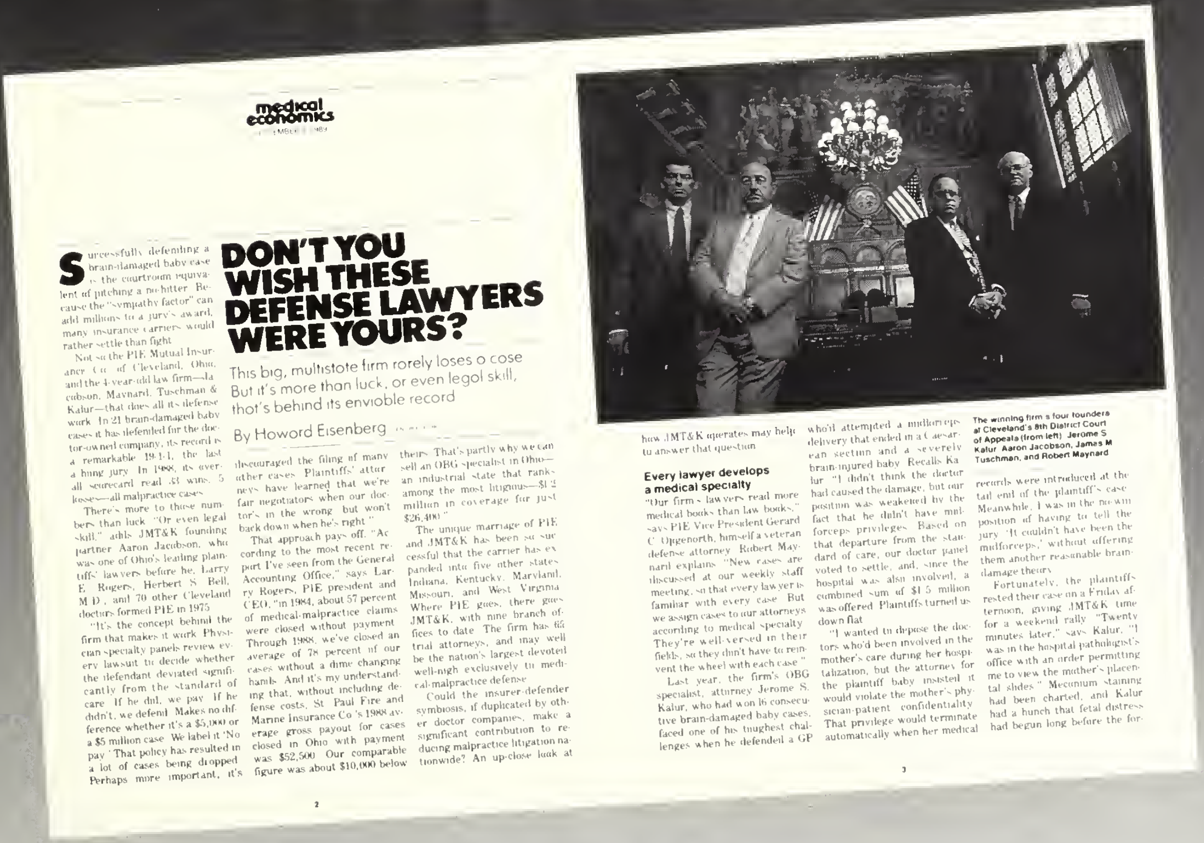

medical economics

DON'T YOU WISH THESE DEFENSE LAWYERS WERE YOURS?

This big, multistate firm rarely loses a case. But it's more than luck, or even legal skill, that's behind its enviable record

By Howard Eisenberg

Successfully defending a brain-damaged baby case is the courtroom equivalent of pitching a no-hitter. Because the "sympathy factor" can add millions to a jury's award, many insurance carriers would rather settle than fight.

Not so the PIE Mutual Insurance Co. of Cleveland, Ohio, and the 4-year-old law firm—Jacobson, Maynard, Tuschman & Kalur—that does all its defense work. In 21 brain-damaged baby cases it has defended for the doctor-owned company, its record is a remarkable 19-1-1, the last a hung jury. In 1988, its overall scorecard read 33 wins, 5 losses—all malpractice cases.

There's more to those numbers than luck. "Or even legal skill," adds JMT&K founding partner Aaron Jacobson, who was one of Ohio's leading plaintiffs' lawyers before he, Larry E. Rogers, Herbert S. Bell, M.D., and 70 other Cleveland doctors formed PIE in 1975.

"It's the concept behind the firm that makes it work. Physician specialty panels review every lawsuit to decide whether the defendant deviated significantly from the standard of care. If he did, we pay. If he didn't, we defend. Makes no difference whether it's a $5,000 or a $5 million case. We label it 'No pay.' That policy has resulted in a lot of cases being dropped. Perhaps more important, it's discouraged the filing of many other cases. Plaintiffs' attorneys have learned that we're fair negotiators when our doctor's in the wrong, but won't back down when he's right."

That approach pays off. "According to the most recent report I've seen from the General Accounting Office," says Larry Rogers, PIE president and CEO, "in 1984, about 57 percent of medical-malpractice claims were closed without payment. Through 1988, we've closed an average of 78 percent of our cases without a dime changing hands. And it's my understanding that, without including defense costs, St. Paul Fire and Marine Insurance Co.'s 1988 average gross payout for cases closed in Ohio with payment was $52,500. Our comparable figure was about $10,000 below theirs. That's partly why we can sell an OBG specialist in Ohio—an industrial state that ranks among the most litigious—$1.2 million in coverage for just $26,400."

The unique marriage of PIE and JMT&K has been so successful that the carrier has expanded into five other states: Indiana, Kentucky, Maryland, Missouri, and West Virginia. Where PIE goes, there goes JMT&K, with nine branch offices to date. The firm has [illegible] trial attorneys, and may well be the nation's largest devoted well-nigh exclusively to medical-malpractice defense.

Could the insurer-defender symbiosis, if duplicated by other doctor companies, make a significant contribution to reducing malpractice litigation nationwide? An up-close look at how JMT&K operates may help to answer that question.

The winning firm's four founders at Cleveland's 8th District Court of Appeals (from left): Jerome S. Kalur, Aaron Jacobson, James M. Tuschman, and Robert Maynard

Every lawyer develops a medical specialty

"Our firm's lawyers read more medical books than law books," says PIE Vice President Gerard C. Opgenorth, himself a veteran defense attorney. Robert Maynard explains: "New cases are discussed at our weekly staff meeting, so that every lawyer is familiar with every case. But we assign cases to our attorneys according to medical specialty. They're well-versed in their fields, so they don't have to reinvent the wheel with each case."

Last year, the firm's OBG specialist, attorney Jerome S. Kalur, who had won 16 consecutive brain-damaged baby cases, faced one of his toughest challenges when he defended a GP who'd attempted a midforceps delivery that ended in a Caesarean section and a severely brain-injured baby. Recalls Kalur: "I didn't think the doctor had caused the damage, but our position was weakened by the fact that he didn't have midforceps privileges. Based on that departure from the standard of care, our doctor panel voted to settle, and, since the hospital was also involved, a combined sum of $1.5 million was offered. Plaintiffs turned us down flat.

"I wanted to depose the doctors who'd been involved in the mother's care during her hospitalization, but the attorney for the plaintiff baby insisted it would violate the mother's physician-patient confidentiality. That privilege would terminate automatically when her medical records were introduced at the tail end of the plaintiff's case. Meanwhile, I was in the no-win position of having to tell the jury 'It couldn't have been the midforceps,' without offering them another reasonable brain-damage theory."

Fortunately, the plaintiffs rested their case on a Friday afternoon, giving JMT&K time for a weekend rally. "Twenty minutes later," says Kalur, "I was in the hospital pathologist's office with an order permitting me to view the mother's placental slides." Meconium staining had been charted, and Kalur had a hunch that fetal distress had begun long before the for-

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[†] Constipation, which is easily managed in most patients, is the most commonly reported side effect of Calan SR.

[‡] Verapamil should be administered cautiously to patients with impaired renal function.

References: 1. Data on file, Searle. 2. Edmonds D, Würth JP, Baumgart P, et al. Twenty-four-hour monitoring of blood pressure during calcium antagonist therapy. In: Fleckenstein A, Laragh SH, eds. *Hypertension—the Next Decade: Verapamil in Focus*. New York, NY: Churchill Livingstone; 1987:94-100. 3. Midtbø KA. Effects of long-term verapamil therapy on serum lipids and other metabolic parameters. *Am J Cardiol*. 1990;66:131-151. 4. Fagher B, Henningsen N, Hulthén L, et al. Antihypertensive and renal effects of enalapril and slow-release verapamil in essential hypertension. *Eur J Clin Pharmacol*. 1990;39(suppl 1):S41-S43. 5. Schmieder RE, Messerli FH, Garavaglia GE, et al. Cardiovascular effects of verapamil in patients with essential hypertension. *Circulation*. 1987;75:1030-1036. 6. Midtbø K, Lauve O, Hals O. No metabolic side effects of long-term treatment with verapamil in hypertension. *Angiology*. 1988;39:1025-1029.

BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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Missouri Medicine

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- The Role of Antibiotics in the Management of Acute Exacerbation of Chronic Bronchitis
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Owned and edited since 1904 by the Missouri State Medical Association. *Missouri Medicine*, Journal of the Missouri State Medical Association, is published monthly at 113 Madison, Jefferson City, Missouri 65102. Second-Class postage paid at Jefferson City, Missouri and at additional mailing offices. Postmaster: Send address changes to Missouri State Medical Association, c/o Membership Dept. P.O. Box 1028, Jefferson City, MO 65102.

The subscription price per year for non-members is $20.00. The subscription price for members is included in the annual membership dues. The international subscription price is $40.00. Single copies are $4.00 and the cost of the annual August Directory issue is $15.00.

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Printed by the Ovid Bell Press, Inc., Fulton, Missouri 65251.

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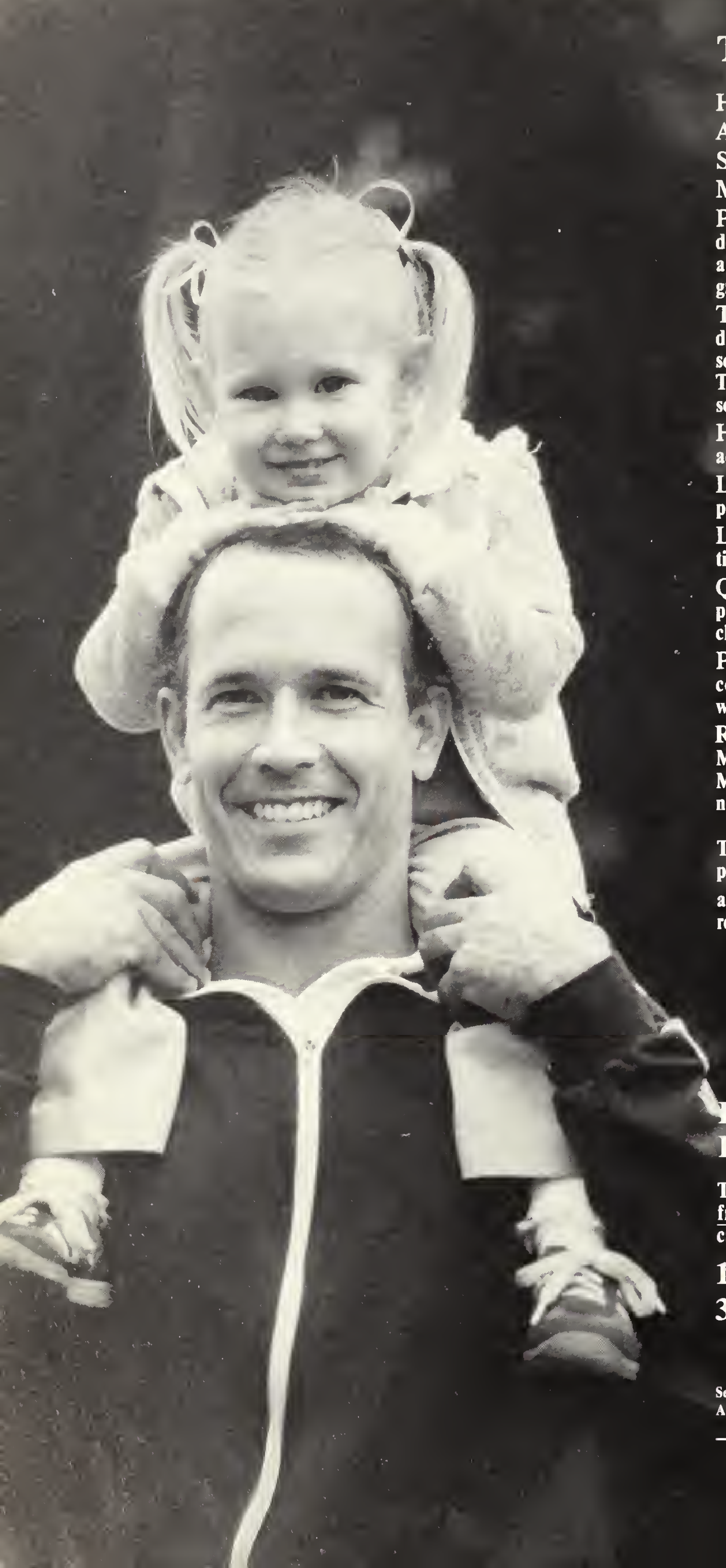

THOMAS P. DOYLE, M.D.

HOME: Springfield, Missouri.

AGE: 36.

SPECIALTY: Orthopedic Surgery.

MEDICAL COLLEGE: University of Missouri, 1978

PRACTICE: "I recently purchased an established but declining sole practitioner office from a retiring physician. It's a risk, but they say that great deeds are usually wrought at great risk."

TYPICAL DAY: "Let the horses out of the corral at dawn and maybe even go for a ride. Then, it's surgery if scheduled and a day of torn ligaments and sprained ankles. The evening finds me on the sidelines of one of our high school sports teams."

HOBBIES: Horseback riding and golf. "If I combined activities, would it be polo?"

LAST BOOK READ: Missouri: Images of Nature, photographed by Charles Gurche.

LATEST ACCOMPLISHMENT: "Testing a protective knee pad I'm developing for use by football players."

QUOTE: "Physicians are being deterred from trying new procedures and therapies in patient care. We need to cut the chains and encourage individual innovation."

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MISSOURI MEDICINE
Journal of the Missouri State Medical Association

Vol. 89, No. 5 May 1992

May's Cover:
This month's cover was done by William J. Taylor, Richmond, Missouri. It is a graphite drawing, 15 1/4" x 19 1/4", and shows a wildlife refuge of the Missouri State Conservation area at Dunksburg, Missouri. Mr. Taylor has won numerous ribbons at the State Fair in Sedalia and the Ozark Empire Fair in Springfield, Missouri.

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Missouri Patient Care Review Foundation
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THE RIGHT MEDICINE

By Dr. John E. Moore, Jr, President of Drury College

The physician as a teacher; a profession based on continued education; both concepts have been fundamental to the tenets and credo of medicine since Hippocrates. Medical education is evolving and changing in response to social pressures (both economic as well as philosophical) certainly as rapidly as the metamorphosis of medical practice that seems to be transpiring.

This is the first in a series of invited editorials addressing medical education in Missouri to be presented through the year. It seems appropriate to begin this sequence by reacquainting ourselves with the motives and aspirations of the young people at the entry level of the profession. In spite of controversy and challenges to medicine in the social-political arena, medical school applications are currently on an upswing after a low point a few years ago. What factors elicit young men and women to pursue medical careers in today's environment?

Dr. John E. Moore, Jr., President of Drury College in Springfield, MO, interacts and counsels these young people on a daily basis. Drury College has a significant history of developing successful pre-medical students attending all four of Missouri's medical schools. His editorial provides insight into the questions and concerns of these young people from a unique perspective.

J. Regan Thomas, M.D., Editor

Dr. Moore

How do young men and women make choices and decisions about career fields? More specifically, what prompts them to choose medicine as a career?

These questions are offered from the perspective of someone who works regularly with students considering medicine as a career. We know that in many cases students have an interest in medicine from the early years. Physicians are strong role models; their work and relationships with patients help shape youngsters' interest in medicine. Academic success in high school reinforces this interest, and as students prepare for college, they begin to think more seriously about becoming doctors.

Our college has a strong and successful program in pre-medicine, preparing ten to fifteen students each year to enter medical school or one of the related health sciences. Their record in medical school has reinforced Drury's fine reputation for pre-medicine. During nine years, I have known personally and worked with many of our pre-med students. My daughter, now a freshman at Drury, is considering a medical and research career.

Each year Drury sponsors a pre-medical information day program for high school students and their parents. The purpose of the program is to provide career information to students who are considering medicine. We believe that this information - ranging from broad ethical issues to "nuts and bolts" practicality - helps students and their families make sound decisions about medicine as a career. Participants in

Continued on page 268

Editor's Desk

Continued from page 267

the pre-medical day program include admissions directors and representatives from the medical and dental schools in Missouri, practicing physicians and dentists, as well as current medical students.

What kind of questions do high school and college students ask about medicine as a career? They range from the philosophical to the practical. Following are representative issues which are of interest to prospective medical students, along with the kinds of responses given.

Question: If I am planning to go to medical school, what should I major in as an undergraduate?

Response: As long as a student takes the recommended science and mathematics courses generally considered as pre-requisites for medical school, the specific major taken as an undergraduate is not crucial. Many medical schools recommend that students take a broad sampling of courses in the liberal arts and consider majors like psychology, history or political science. The breadth and richness of a student's undergraduate education should make him or her a better physician.

Question: What are some of the most important abilities an effective doctor should have?

Response: Practicing physicians, medical students and medical admissions officers seem to agree that strong communication skills (both written and oral), analytical skills and the ability to relate well to people are all quite important. Again, strong liberal arts and general education at the undergraduate level are emphasized besides a strong background in the sciences and medicine.

Question: What satisfaction does medicine offer as a career?

Response: The opportunity to work with people, to have a broad variety of issues and challenges and the chance to earn a comfortable living are often mentioned.

Question: What do medical schools look for in admitting students?

Response: Medical schools generally are looking for the best qualified applicants with a well-rounded combination of aptitude (medical college admissions test scores and recommendations), achievement (college grades and recommendations), breadth of background (general education, special interests) and personal promise (personality, motivation and career goals). State-supported schools usually also give preference to students from the same state. Applicants to medical school have increased in many institutions in recent years, and clearly admission continues to be competitive.

Question: What does a medical Education cost?

Response: While it is expensive, it is possible for a student with motivation and determination. Financial aid and loans are available and students should not be reluctant to take on some debt in completing a medical degree. Special programs like the public health and military services assist selected students in obtaining their medical education. Carefully planned debt should be considered an investment and can be managed. Financial aid advisors in college and medical school can assist in planning to finance a medical education.

It is interesting that questions about doctors' income and the prestige they enjoy as professionals seldom arise. There is widespread acknowledgment that the practice of medicine is changing, and that one of the frustrations confronting doctors is the paperwork and regulation they must manage.

These disadvantages notwithstanding, pre-medical students seem to consider careers in medicine with considerable altruism and commitment. These qualities, together with a solid general education and careful preparation in the sciences, should result in the right medicine for a career as a physician.

Dear Dr. Thomas:

Missouri physicians and the MSMA are expressing concern and increasing criticism of the expanding bureaucracy of the State Board of Registration for the Healing Arts and its avowed policy to increase investigative and disciplinary actions. With apparent disdain to criticism, the licensure fee was increased in 1992 to $120, and the extra funds created used to hire additional investigator agents and finance prosecution costs. Ethical standards have been cast aside, protests by the MSMA have received little attention, and the situation has worsened. The executive director of the state board now designates it a "Consumer Protection Board," and states the need to investigate all consumer complaints. There is, and continues to be, a market increase in reported cases with a backlog of over 1100 cases, with the majority reported as being based on consumer complaints.

A reasonable question is now being asked as to who is protecting the physicians from prolonged hearings and unrealistic rulings because of this regulatory activity. The answer is not in the simple statement that "you need a lawyer" as proclaimed in a large advertisement in recent issues of the *Missouri Medicine* that cites the fact that "There are 1100 physicians under investigation by the Missouri Board of Healing Arts." This may be a lawyer's mecca, but it is a catastrophe to the medical profession. I agree with Dr. Arthur H. Gale in his special article in the January 1992 issue of *Missouri Medicine*; that the State Board has gone too far with their unjustified actions.

I have noted significant changes that have been made by the State Board in both the appearance and in the wording on the 1991 registration and/or license certificate. The format of the colorful border, and the gold embossed prominently displayed "State of Missouri" across the top portion is beautiful. But my concern, to which I direct attention, is in the wording compared to previous certificates. The 1991 certificate reads, "and is hereby authorized to engage in the activity as Physician and Surgeon," whereas the 1992 certificate reads, "and is hereby authorized to be registered and/or licensed as Physician and Surgeon." As I interpret the change in wording, a physician is registered, but is not authorized to practice by the 1992 certificate.

It is illogical to assume that the increased investigative and disciplinary activity by the State Board of Healing Arts will improve the quality of care or the availability of care in the state. Many general practitioners I know have retired rather than cope with regulations and harassment by the investigative agents. There are no provisions for a so-called twilight practice in Missouri; and under the current regulation a retired physician cannot engage in the practice of medicine in any form or fashion including occasional or gratuitous practice. The recently introduced "Good Samaritan Bill," to allow gratuitous practice by retired physicians, failed to pass. The wasteful misuse of medical resources continues.

I practiced medicine in rural north Missouri for over 50 years, but in 1990 I had to accept "forced" retirement to retain and continue licensure. I did not want to retire. I was not disabled physically, and had complied with Missouri statutes as to licensure. But retirement can be, and is used by the State Board as a punitive measure; and as I had signed a retirement agreement all efforts for re-entry, even for limited practice, were futile. My attorney says my license is useless, but I consider my license as a definite expression of my qualifications, even though it authorizes no activity. I also consider the license as a link to organized medicine which has prompted and ensured the finest medical care in the world. I deplore injustice and tyranny and served on active duty with the USAF in three wars to secure freedom for the oppressed.

John R. Dixon, M.D., Brookfield, MO

Dear Editor:

I am writing to reach physicians regarding tuberculosis control and elimination activities. Specifically, physicians need to know that tuberculous infection (i.e., a positive skin test) is now a reportable condition in Missouri. Tuberculous infection became a reportable condition as of March 14, 1991. It is a Department of Health rule that is listed in 19 CSR 20-20.020. However, many physicians still are unaware that tuberculous infection is reportable.

The reason that tuberculous infection became a reportable condition is that we are interested in preventing future cases of tuberculosis. The reporting mechanism and follow-up will enable the public and private sectors to work together to ensure that our patients receive the appropriate preventive therapy. As a result of these actions, we hope to prevent future cases of active tuberculosis and to eliminate this disease in Missouri by the year 2010. As you know, tuberculosis is resurging and is no longer only a disease of the past.

Call your local health department or the Bureau of Tuberculosis Control at 314/751-6122 for additional information.

Vic Tomlinson, Bureau of Tuberculosis Control

Poetry by S.D. Madduri, M.D.

A PHYSICIAN'S PRAYER

This is my prayer to thee my Lord,
A prayer from the heart and soul of a disenchanted physician
Who is trying to practice medicine under adverse conditions
And who is trying to calm his conscience with hope for future.

Medical practice ain't any more what it used to be
Nor it ain't what it is intended to be
It is not to heal the suffering and to mend the sick
Medicine is an "industry" with no heart or feelings
It is a freeway for giant corporations to satisfy their greed
It is the victim of malpractice crunch.

So, lead me into a world My Lord, where:
I can go back to practicing medicine like our forefathers did
I can spend my time healing the suffering
But not marketing my expertise
I don't have to order an extra lab test to save my 'skin'
I don't have to look into the 'criteria' before I admit the sick
Nor do I have to release him because his 'D.R.G.s' are up.

Make this world worthy of living, My lord,
Where the sun is not afraid to shine
Where people are not afraid to get sick
And where medicine could be practiced as 'Medicine.'

LONG, EMPTY HALLWAYS

As I stood at the long, empty hallway
As I walked through those deserted aisles
And heard the deadly silence that prevailed
Remembered those empty conduits
Are the life lines in a busy general hospital.

Come by morning, I know
The hallways will bustle with life
They witness ups and downs of human drama
The agony of sickness and suffering
The relentless cries of children in pain
Tears tinged with blood from loss of loved ones
Last glow in the sunken eyes of cancer patients.

Strange may it be
The same hallways stand witness to
Joy of suffering souls relieved of pain and sickness
Proud look of parents taking their new baby home
Smile that lit the young lady's face
That her breast mass was not cancer.

As I stood back and watched
I know those empty hallways
Those long, endless, white walls
The chaotic, cluttering signs
And the tasteless murals on the walls
Will echo with life and death again.

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New!

ONCE-A-DAY ‡

ACCUPRIL® ™

quinapril HCl tablets 10, 20, 40 mg

* See DOSAGE AND ADMINISTRATION section of prescribing information.

† If, after an adequate trial of ACCUPRIL alone, based on your medical judgment as the prescribing physician, you determine that your patient requires the addition of a diuretic, Parke-Davis will refund to the patient his/her cost for the diuretic prescription less any amount reimbursed or paid for by an HMO, insurance company, or any other plan or program.

For more details, ask your Parke-Davis Representative or call 1-800-955-3077.

‡ In some patients, the antihypertensive effect may diminish toward the end of the once-daily dosing interval. In such patients, an increase in dosage or twice-daily administration may be warranted.

ACCUPRIL is available in 10, 20, and 40 mg tablets. Usual initial starting dosage is 10 mg once daily.

ACCUPRIL is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Please see brief summary of prescribing information on following page.

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PARKE-DAVIS

Accupril® (Quinapril Hydrochloride Tablets)

Before prescribing, please see full prescribing information. A brief summary follows.

INDICATIONS AND USAGE

ACCUPRIL is indicated for the treatment of hypertension. It may be used alone or in combination with thiazide diuretics.

In using ACCUPRIL, consideration should be given to the fact that another angiotensin-converting enzyme (ACE) inhibitor, captopril, has caused agranulocytosis, particularly in patients with renal impairment or collagen vascular disease. Available data are insufficient to show that ACCUPRIL does not have a similar risk (see WARNINGS).

CONTRAINDICATIONS

ACCUPRIL is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

WARNINGS

Angioedema: Angioedema of the face, extremities, lips, tongue, glottis, and larynx has been reported in patients treated with ACE inhibitors and has been seen in 0.1% of patients receiving ACCUPRIL. Angioedema associated with laryngeal edema can be fatal. If laryngeal stridor or angioedema of the face, tongue, or glottis occurs, treatment with ACCUPRIL should be discontinued immediately, the patient treated in accordance with accepted medical care, and carefully observed until the swelling disappears In instances where swelling is confined to the face and lips, the condition generally resolves without treatment, antihistamines may be useful in relieving symptoms.

Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, emergency therapy including, but not limited to, subcutaneous epinephrine solution 1:1000 (0.3 to 0.5 mL) should be promptly administered (see ADVERSE REACTIONS).

Hypotension: Symptomatic hypotension was rarely seen in uncomplicated hypertensive patients treated with ACCUPRIL but, as with other ACE inhibitors, it is a possible consequence of therapy in salt/volume depleted patients, such as those previously treated with diuretics or dietary salt restriction or who are on dialysis (see PRECAUTIONS, DRUG INTERACTIONS, and ADVERSE REACTIONS). In controlled studies, syncope was observed in 0 4% of patients (N = 3203); this incidence was similar to that observed for captopril (1%) and enalapril (0.8%).

In patients with concomitant congestive heart failure, with or without associated renal insufficiency, ACE inhibitor therapy may cause excessive hypotension, which may be associated with oliguria or azotemia and, rarely, with acute renal failure and death. In such patients, ACCUPRIL therapy should be started at the recommended dose under close medical supervision. These patients should be followed closely for the first 2 weeks of treatment and whenever the dosage of antihypertensive medication is increased (see DOSAGE AND ADMINISTRATION).

If symptomatic hypotension occurs, the patient should be placed in the supine position and, if necessary, normal saline may be administered intravenously. A transient hypotensive response is not a contraindication to further doses; however, lower doses of ACCUPRIL or reduced concomitant diuretic therapy should be considered

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression rarely in patients with uncomplicated hypertension, but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease such as systemic lupus erythematosus or scleroderma. Agranulocytosis did occur during ACCUPRIL treatment in one patient with a history of neutropenia during previous captopril therapy. Available data from clinical trials of ACCUPRIL are insufficient to show that, in patients without prior reactions to other ACE inhibitors, ACCUPRIL does not cause agranulocytosis at similar rates. As with other ACE inhibitors, periodic monitoring of white blood cell counts in patients with collagen vascular disease and/or renal disease should be considered.

Fetal/Neonatal morbidity and mortality: ACE inhibitors, including ACCUPRIL, can cause fetal and neonatal morbidity and mortality when administered to pregnant women.

When ACE inhibitors have been used during the second and third trimesters of pregnancy, there have been reports of hypotension, renal failure, skull hypoplasia, and death. Oligohydramnios has also been reported, presumably resulting from decreased fetal renal function; oligohydramnios has been associated with fetal limb contractures, craniofacial deformities, hypoplastic lung development, and intrauterine growth retardation

Prematurity and patent ductus arteriosus have been reported, although it is not clear whether these occurrences were due to the ACE-inhibitor exposure or to the mother's underlying disease. It is not known whether exposure limited to the first trimester can adversely affect fetal outcome.

New!

ONCE-A-DAY*

ACCUPRIL® ™

quinapril HCl tablets

A patient who becomes pregnant while taking ACE inhibitors, or who takes ACE inhibitors when already pregnant, should be apprised of the potential hazard to her fetus. If she continues to receive ACE inhibitors during the second or third trimester of pregnancy, frequent ultrasound examinations should be performed to look for oligohydramnios. When oligohydramnios is found, ACE inhibitors should generally be discontinued.

Infants with histories of in utero exposure to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion. Hemodialysis and peritoneal dialysis have little effect on the elimination of quinapril and quinaprilat

No fetotoxic or teratogenic effects were observed in rats at quinapril doses as high as 300 mg/kg/day (180 and 30 times the maximum daily human dose when based on mg/kg and mg/m^2, respectively), despite maternal toxicity at 150 mg/kg/day Tested later in gestation and during lactation, reduced offspring body weight was seen at ≥25 mg/kg/day, and changes in renal histology (juxtaglomerular cell hypertrophy, tubular/pelvic dilation, glomerulosclerosis) were observed both in dams and offspring treated with 150 mg/kg/day. Quinapril was not teratogenic in the rabbit, however, as noted with other ACE inhibitors, maternal toxicity and embryotoxicity were seen in some rabbits at quinapril doses as low as 0.5 mg/kg/day (one time the recommended human dose) and 1.0 mg/kg/day, respectively

PRECAUTIONS

General

Impaired renal function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including ACCUPRIL, may be associated with oliguria and/or progressive azotemia and rarely acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine have been observed in some patients following ACE inhibitor therapy. These increases were almost always reversible upon discontinuation of the ACE inhibitor and/or diuretic therapy In such patients, renal function should be monitored during the first few weeks of therapy.

Some hypertensive patients with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when ACCUPRIL has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of any diuretic and/or ACCUPRIL may be required

Evaluation of hypertensive patients should always include assessment of renal function (see DOSAGE AND ADMINISTRATION).

Hyperkalemia and potassium-sparing diuretics: In clinical trials, hyperkalemia (serum potassium ≥5.8 mmol/L) occurred in approximately 2% of patients receiving ACCUPRIL. In most cases, elevated serum potassium levels were isolated values which resolved despite continued therapy. Less than 0.1% of patients discontinued therapy due to hyperkalemia Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with ACCUPRIL (see PRECAUTIONS, Drug Interactions).

Surgery/anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, ACCUPRIL will block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients

Angioedema: Angioedema, including laryngeal edema, can occur with treatment with ACE inhibitors, especially following the first dose. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to stop taking the drug until they have consulted with their physician (see WARNINGS).

Symptomatic hypotension: Patients should be cautioned that lightheadedness can occur, especially during the first few days of ACCUPRIL therapy, and that it should be reported to a physician. If actual syncope occurs, patients should be told to not take the drug until they have consulted with their physician (see WARNINGS).

All patients should be cautioned that inadequate fluid intake or excessive perspiration, diarrhea, or vomiting can lead to an excessive fall in blood pressure because of reduction in fluid volume, with the same consequences of lightheadedness and possible syncope

Patients planning to undergo any surgery and/or anesthesia should be told to inform their physician that they are taking an ACE inhibitor.

Hyperkalemia: Patients should be told not to use potassium supplements or salt substitutes containing potassium without consulting their physician (see PRECAUTIONS).

Accupril® (Quinapril Hydrochloride Tablets)

Neutropenia: Patients should be told to report promptly any indication of infection (eg, sore throat, fever) which could be a sign of neutropenia

NOTE As with many other drugs, certain advice to patients being treated with ACCUPRIL is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions

Concomitant diuretic therapy: As with other ACE inhibitors, patients on diuretics, especially those on recently instituted diuretic therapy, may occasionally experience an excessive reduction of blood pressure after initiation of therapy with ACCUPRIL The possibility of hypotensive effects with ACCUPRIL may be minimized by either discontinuing the diuretic or cautiously increasing salt intake prior to initiation of treatment with ACCUPRIL If it is not possible to discontinue the diuretic, the starting dose of quinapril should be reduced (see DOSAGE AND ADMINISTRATION).

Agents increasing serum potassium: Quinapril can attenuate potassium loss caused by thiazide diuretics and increase serum potassium when used alone. If concomitant therapy of ACCUPRIL with potassium-sparing diuretics (eg, spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes is indicated, they should be used with caution along with appropriate monitoring of serum potassium (see PRECAUTIONS).

Tetracycline and other drugs that interact with magnesium: Simultaneous administration of tetracycline with ACCUPRIL reduced the absorption of tetracycline by approximately 28% to 37%, possibly due to the high magnesium content in ACCUPRIL tablets. This interaction should be considered if coprescribing ACCUPRIL and tetracycline or other drugs that interact with magnesium.

Lithium: Increased serum lithium levels and symptoms of lithium toxicity have been reported in patients receiving concomitant lithium and ACE inhibitor therapy These drugs should be co-administered with caution, and frequent monitoring of serum lithium levels is recommended. If a diuretic is also used, it may increase the risk of lithium toxicity.

Other agents: Drug interaction studies of ACCUPRIL with other agents showed

- Multiple dose therapy with propranolol or cimetidine has no effect on the pharmacokinetics of single doses of ACCUPRIL.
- The anticoagulant effect of a single dose of warfarin (measured by prothrombin time) was not significantly changed by quinapril coadministration twice-daily
- ACCUPRIL treatment did not affect the pharmacokinetics of digoxin.
- No pharmacokinetic interaction was observed when single doses of ACCUPRIL and hydrochlorothiazide were administered concomitantly

Carcinogenesis, Mutagenesis, Impairment of Fertility

Quinapril hydrochloride was not carcinogenic in mice or rats when given in doses up to 75 or 100 mg/kg/day (50 to 60 times the maximum human daily dose, respectively, on a mg/kg basis and 3.8 to 10 times the maximum human daily dose when based on a mg/m^2 basis) for 104 weeks. Female rats given the highest dose level had an increased incidence of mesenteric lymph node hemangiomas and skin/subcutaneous lipomas. Neither quinapril nor quinaprilat were mutagenic in the Ames bacterial assay with or without metabolic activation. Quinapril was also negative in the following genetic toxicology studies: *in vitro* mammalian cell point mutation, sister chromatid exchange in cultured mammalian cells, micronucleus test with mice, *in vitro chromosome* aberration with V79 cultured lung cells, and in an *in vivo* cytogenetic study with rat bone marrow There were no adverse effects on fertility or reproduction in rats at doses up to 100 mg/kg/day (60 and 10 times the maximum daily human dose when based on mg/kg and mg/m^2, respectively).

Pregnancy

Pregnancy Category D: See WARNINGS, Fetal/Neonatal morbidity and mortality.

Nursing Mothers

It is not known if quinapril or its metabolites are secreted in human milk Quinapril is secreted to a limited extent, however, in milk of lactating rats (5% or less of the plasma drug concentration was found in rat milk). Because many drugs are secreted in human milk, caution should be exercised when ACCUPRIL is given to a nursing mother.

Geriatric Use

Elderly patients exhibited increased area under the plasma concentration time curve (AUC) and peak levels for quinaprilat compared to values observed in younger patients; this appeared to relate to decreased renal function rather than to age itself. In controlled and uncontrolled studies of ACCUPRIL where 918 (21%) patients were 65 years and older, no overall differences in effectiveness or safety were observed between older and younger patients. However, greater sensitivity of some older individual patients cannot be ruled out.

Pediatric Use

The safety and effectiveness of ACCUPRIL in children have not been established

ADVERSE REACTIONS

ACCUPRIL has been evaluated for safety in 4960 subjects and patients. Of these, 3203 patients, including 655 elderly patients, participated in controlled clinical trials. ACCUPRIL has been evaluated for long-term safety in over 1400 patients treated for 1 year or more.

Adverse experiences were usually mild and transient.

Discontinuation of therapy because of adverse events was required in 4.7% of patients treated with ACCUPRIL in placebo-controlled hypertension trials

Adverse experiences probably or possibly related to therapy or of unknown relationship to therapy occurring in 1% or more of the 1563 patients in placebo-controlled hypertension trials who were treated with ACCUPRIL are shown below.

Adverse Events in Placebo-Controlled Trials

	ACCUPRIL (N = 1563) Incidence (Discontinuance)	Placebo (N = 579) Incidence (Discontinuance)
Headache	5.6 (0.7)	10.9 (0.7)
Dizziness	3.9 (0.8)	2.6 (0.2)
Fatigue	2.6 (0.3)	1.0
Coughing	2 0 (0.5)	0.0
Nausea/Vomiting	1.4 (0 3)	1.9 (0.2)
Abdominal Pain	1.0 (0.2)	0.7

Clinical adverse experiences probably or possibly related, or of uncertain relationship to therapy, occurring in 0.5% to 1.0% (except as noted) of the patients treated with ACCUPRIL (with or without concomitant diuretic) in controlled or uncontrolled trials (N = 4397) and less frequent, clinically significant events seen in clinical trials or post-marketing experience (the rarer events are in italics) include (listed by body system):

General: back pain, malaise

Cardiovascular: palpitation, vasodilation, tachycardia, *heart failure*, *hyperkalemia*, *myocardial infarction*, *cerebrovascular accident*, *hypertensive crisis*, *angina pectoris*, *orthostatic hypotension*, *cardiac rhythm disturbances*

Gastrointestinal: dry mouth or throat, constipation, *gastrointestinal hemorrhage*, *pancreatitis*, *abnormal liver function tests*

Nervous/Psychiatric: somnolence, vertigo, syncope, *nervousness*, *depression*

Integumentary: increased sweating, pruritus, *exfoliative dermatitis*, *photosensitivity reaction*

Urogenital: *acute renal failure*

Other: amblyopia, pharyngitis, sinusitis, bronchitis, *agranulocytosis*, *thrombocytopenia*

Angioedema: angioedema has been reported in patients receiving ACCUPRIL (0. 1%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with ACCUPRIL should be discontinued and appropriate therapy instituted immediately (See WARNINGS.)

Clinical Laboratory Test Findings

Hematology: (See WARNINGS)

Hyperkalemia: (See PRECAUTIONS)

Creatinine and blood urea nitrogen: Increases (>1.25 times the upper limit of normal) in serum creatinine and blood urea nitrogen were observed in 2% and 2%, respectively, of patients treated with ACCUPRIL alone. Increases are more likely to occur in patients receiving concomitant diuretic therapy than in those on ACCUPRIL alone. These increases often remit on continued therapy

* In some patients, the antihypertensive effect may diminish toward the end of the once-daily dosing interval. In such patients, an increase in dosage or twice-daily administration may be warranted.

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A VIEW FROM THE WINDOW

by

Carol Williams, M.D.
President, Missouri State Medical Association

Carol Williams, M.D.

After the inaugural, after the reception and greeting old friends and new, after the quiet celebration with family and friends, I sat in the window on the fifteenth floor of the hotel and absorbed the sights and sounds of the city of night: the plaintive hook of the Spirit of St. Louis as she came upstream and docked at the levee, the stream of cars across the Poplar Street Bridge, the known but unseen trains crossing the Eads Bridge and entering the tunnel in front of the arch, planes approaching Lambert Field, the clop-clop of hooves drawing carriages along the streets of St. Louis, a long barge going by on the river. What a richness and diversity of transportation was going by my window. All this was occurring at a time when Earth was renewing herself in a patch of earth under my window - tulips, trees leafing-out and grass turning green. There was the juxtaposition of ancient faith in the Old Cathedral and not-so-ancient injustice in the Old Courthouse on whose steps the Dred Scott decision was handed down.

Across Missouri various county physicians have begun devising new and reviewing old ways of providing health care to the medically needy. They are doing this without waiting for the full development of either a state or national health care plan. In some situations there are free clinics staffed by physicians. In others physicians are backup for certified nurse midwives and nurse practitioners working under clearly established protocols. In some counties physicians take turns providing care for the indigent who drop into emergency rooms. Throughout the state physicians provide care to the medically indigent in their offices.

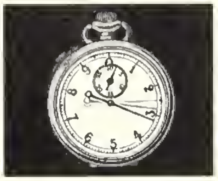
Longterm solutions to problems of access are being sought by consorting of business, labor, insurance, medical, religious, and political figures in the metropolitan areas.

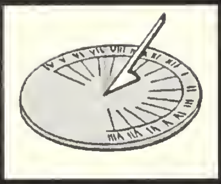
All of these efforts are clear signs that both shortterm and longterm remedies for a beleaguered health care system are both in place and being searched for. The diversity of their efforts means that like the multiple modes of transportation seen from a hotel window one night - some old, some new - what emerges from the efforts to heal the health care system will probably vary from community to community and will be flexible enough to accommodate the different needs of different areas of the state.

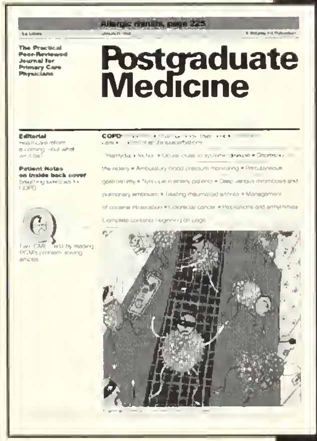
Our role as physicians is to know the injustices of the current system and to bring to the conference table our conviction about the need to preserve those things in the health care scene that must be retained and to change those things which should be changed.

Dr. Williams was inagurated as MSMA's president April 11, 1992, at the Adam's Mark Hotel, St Louis, during the Missouri State Medical Association's 134th Annual Convention.

US **THEM**







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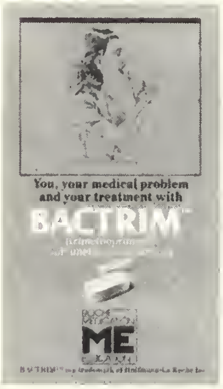
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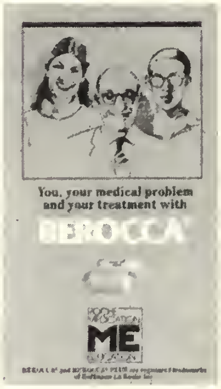
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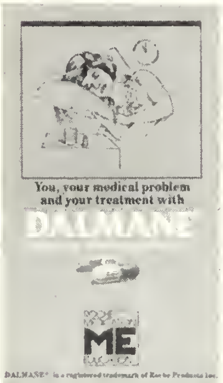
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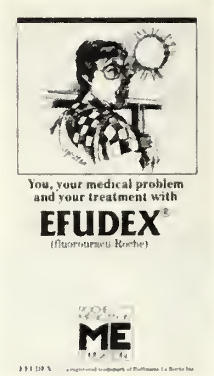
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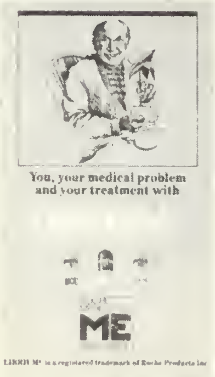
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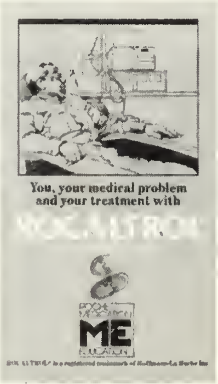
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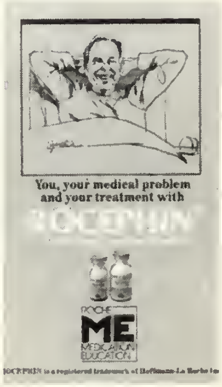
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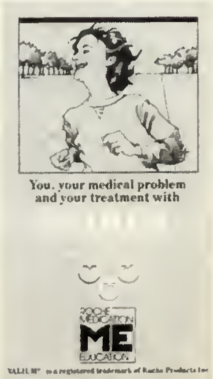
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Dear Dr. Woerth,
There is some confusion in our surgery office as to how to use modifier 50 and 51. Could you please explain?

K.K.

Dear K.K.,

The bilateral procedures modifier (50) is used, according to the CPT book, whenever bilateral procedures are performed in the same operative session. Because the definition of the modifier refers to the operative session, it is assumed that the modifier can only be used for surgical procedures (which includes diagnostic procedures found in the surgery section of CPT). The only other section of the CPT books that it might be appropriate to use the bilateral modifier is with radiology codes. But a check of the modifiers located in the guidelines of the radiology section of CPT finds that 50 is not included in the commonly used modifiers. This does not mean that the bilateral modifier is not to be used with radiology codes. More commonly, when bilateral x-rays are taken, a 2 is marked on the claim form in the days/units box on the new HCFA 1500 to indicate two units of the x-rays were taken.

When using the bilateral modifier for bilateral surgery or diagnostic procedures, add 50 to the CPT code, thus indicating that the procedure was bilateral.

The multiple procedures modifier (51) is used when multiple procedures are performed on the same day or in the same session. The modifier is added to the secondary or lesser procedure. Because many insurance companies pay 50 percent for the second procedure performed in the operative session or on the same day, be sure that the secondary/lesser procedure listed with the modifier 51 is the least expensive of the two procedures. Likewise, when more than two procedures are performed in the same session or on the same day, place the procedures on the claim in descending dollar value as many insurance companies pay only 25 percent for the third and subsequent procedures. The modifier 51 is used on all multiple procedures listed, i.e. the second, third, fourth, etc..

Dear Dr. Woerth,
I understand that the definition of global surgery for Medicare has been changed. What is it and how is it different from that listed in the CPT book?

Dr. P.W.

Dear Dr. P.W.,

Medicare has indeed changed the definition of global surgery. It is as follows: major surgery includes the intra-operative services that are a necessary and usual part of the surgery, all pre-operative visits performed in or out of the hospital that are performed one day prior to the surgery, and all post-operative services for 90 days that are related to the surgery that do not require a trip back to the operating room. This is important. The initial evaluation or consultation by the surgeon will be paid separately from the global surgery package. This is something new for most Medicare carriers as this initial evaluation to determine surgery is needed has generally been part of the global surgery package.

The definition for global surgery that is listed in the CPT book is quite different from the Medicare definition. The CPT book in guideline 2 of the Surgery section states that the surgical package includes the operation, local, topical or block anesthesia and the normal, uncomplicated follow-up care. Thus for the insurance carriers following the CPT book, you should charge separately for all pre-operative services and for any post-operative complications.

There is obviously considerable difference between the Medicare definition of global surgery and that of the CPT book. Be careful that patients are billed properly for pre-operative and post-operative surgical services. Failure to do so could cost an office potentially thousands of dollars in lost revenue. ■

Do you have a question for Dr. Woerth? Include your questions along with your name, address and phone number and send them to:

Janice K. Woerth, Ph.D.
Practice Management
c/o *Missouri Medicine*
P.O. Box 1028
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Although your name will not be used in *Missouri Medicine,* please include your name and phone number so that Dr. Woerth may contact you if she needs additional information to answer your question.

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MARGE PERKINS, THE 1992-1993 AUXILIARY PRESIDENT

By
David E. Perkins, M.D.

This year is an election year and the country is being deluged with information about who the various candidates are, their accomplishments and their positions on the issues facing the country.

I would like to tell you about the 1992-1993 President of the Missouri State Medical Association Auxiliary and her accomplishments, leadership abilities and experiences. The new president is Marge Perkins from the St. Louis Metropolitan Medical Society Auxiliary.

If you look back to the byline of this article, you will see that the new MSMA Auxiliary president is my wife. While this fact would lead you to believe that this article is biased (true), let me assure you that no one else has a more complete knowledge of Marge's background and her many accomplishments, as well as the leadership abilities that she brings to the office of president of the MSMAA.

Marge and I have known each other since age three, when we were in the same Sunday School class. We began to date our senior year at Southwest High School in St. Louis, MO. After one year at Lindenwood College, she transferred to Washington University in St. Louis, where we both graduated, Marge with an AB majoring in English, Speech and Drama. During those years Marge became a Girl Scout leader and graduated from, and became a leader in the American Youth Foundation Leadership Training at Camp Miniwanca, MI.

While I went to medical school at Washington University and the internship and radiology residency in St. Louis, she taught both high school and elementary school. We were married after my freshman year of medical school.

After residency it was on to the United States Air Force, first at Shaw AFB, SC, and Elemdorf AFB in Anchorage, AK. During these years, Marge became active in the Officer's Wives Clubs. While at Elmendorf AFB, she was appointed editor of *"Totem Topics"*, an officer's wives magazine that sold more advertising any of the other magazines. Incidentally, Marge and I both survived the great Alaskan Earthquake, which measured an awesome 8.7 on the Richter scale.

After my Air Force days, we returned to St. Louis and I joined the Scott Radiological Group. Marge became an active volunteer in many organizations. These include the St. Louis Zoo Gift Shop, as a salesperson, several committee chairs for the Volunteer Association of the St. Louis Symphony and a guide at Tower Grove House in the Missouri Botanical Garden. She is also a Guarantor of the Muny Opera, a member of the St. Louis Art, Science and History Museums and a Green Room member of the St. Louis Symphony.

Marge held most of the offices in the Medical Auxiliary of Lutheran Medical Center, including two years as president. She has served on many committees and held many offices in the St. Louis Metropolitan Medical Society Auxiliary, including being its president. She was the chairman of the Gala Committee when the St. Louis Metropolitan Medical Society was 150 years old.

Marge enjoys swimming, bowling, traveling, entertaining and especially golf, which she has chosen for her theme for the coming year - "GO FOR(E) IT...MSMAA." She has been active in the Women's Golf Association at Greenbrier Hills Country Club and served as its president for two years.

Marge's talents for organization and leadership has continued on in the MSMAA. As membership vice president, she led a most successful membership drive. She served as vice president for Health Projects for two years, as well as secretary of the MSMAA. She won the Jean Duensing Journalism Award from the MSMAA in 1989.

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AUXILIARY PAGE

Continued from page 277

Last spring Marge was elected president-elect of the MSMAA and now becomes its president. In doing so, Marge follows a long list of talented previous presidents of the Auxiliary, many of whom I have personally met and have come to admire.

Marge strongly believes in the Auxiliary being a stalwart supporter of the Missouri State Medical Association and its membership. This support starts with a strong, viable organization and Marge stresses the need for increased membership in the MSMA Auxiliary. The many and valuable health projects will continue and can help bring the views of medicine to the attention of the public.

Marge will also stress increasing political activism to help the MSMA and MSMAA let our elected public officials know where medicine stands on the crucial issues now being considered on both the state and national levels.

There is no doubt that Marge Perkins has the dedication, experience, organizational and leadership abilities, as well as the vision to effectively carry out the objectives of the Missouri State Medical Association Auxiliary.

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A NEW MEDICAL GOAL FOR
A SOCIETY OF INDIVIDUALS

"DOCTOR, I WANT . . ."

By Dennis Weiser, Managing Editor

We still can't agree on what caused the social revolution that transformed America's culture 30 years ago. There is unanimity, however, that the "civil rights movement" of the 1960s radically rattled every strata of our society. Even in the 1990s, the aftershocks agitate our social paradigms as we rebuild our fractured society in ways acceptable to a nation of 240 million people who each want to be treated as individuals.

The movement, actually a mélange of social agenda, erupted in numerous countries, but flourished most successfully in America. "Power to the people" opened wide the possibilities about what it means to be a member of a minority, a woman, a senior citizen, or a gay person in American society. It changed how people think about themselves: what they can reasonably expect as persons from society and what obligations, if any, are due in return to society. Persons seized control of their lives, even at the risk of making choices that may not be in their own best interests.[1] [2]

During the early part of the 20th century in America, the medical profession was a good place to find rugged individualists. A professional life that included treating neighbors at four o'clock in the morning armed only with whatever remedies were packed in the bottom of a black bag demanded personal strength. A patient's trust in a physician's power to heal had as much to do with curing illness in that day as the scant medical technology that was available.

By the 1950s, the image of the feisty, much loved, family practitioner as "ideal doctor" had been replaced by a different breed of doctors claims Eric J. Cassell, MD. Medical practitioners, at least in the public mind, were more likely to be thought of as wealthy, prestigious, and authoritative professionals first and as healers second. A medical ethicist, Cassell writes about how and why the dynamics between physicians, society and medicine have changed during his life time in *The Nature of Suffering and The Goals of Medicine.* [3]

The changing perception of the physician was one of the consequences of the profession's shift away from clinical (anecdotal) medicine to an academic, scientific style. Disease-based diagnosis and treatment began to dominate the professional approach to sick persons. Personal feelings and concerns, in the context of the patient/physician relationship, did not add anything to the physician's ability to identify the disease or treat the body. Healing lost peace of mind or easing sick souls was left to others. A friendly "bedside manner" didn't hurt, nor did it "help" cure the disease. This strategy produced astoundingly excellent technical results.

Unhappily, this approach also produced hi-tech physicians whose concern with technology and knowledge of the body reduces sensitivity to a patient's emotional or human-value needs. Children of the 60s grumble because they want hi-touch and hi-tech, not one or the other.

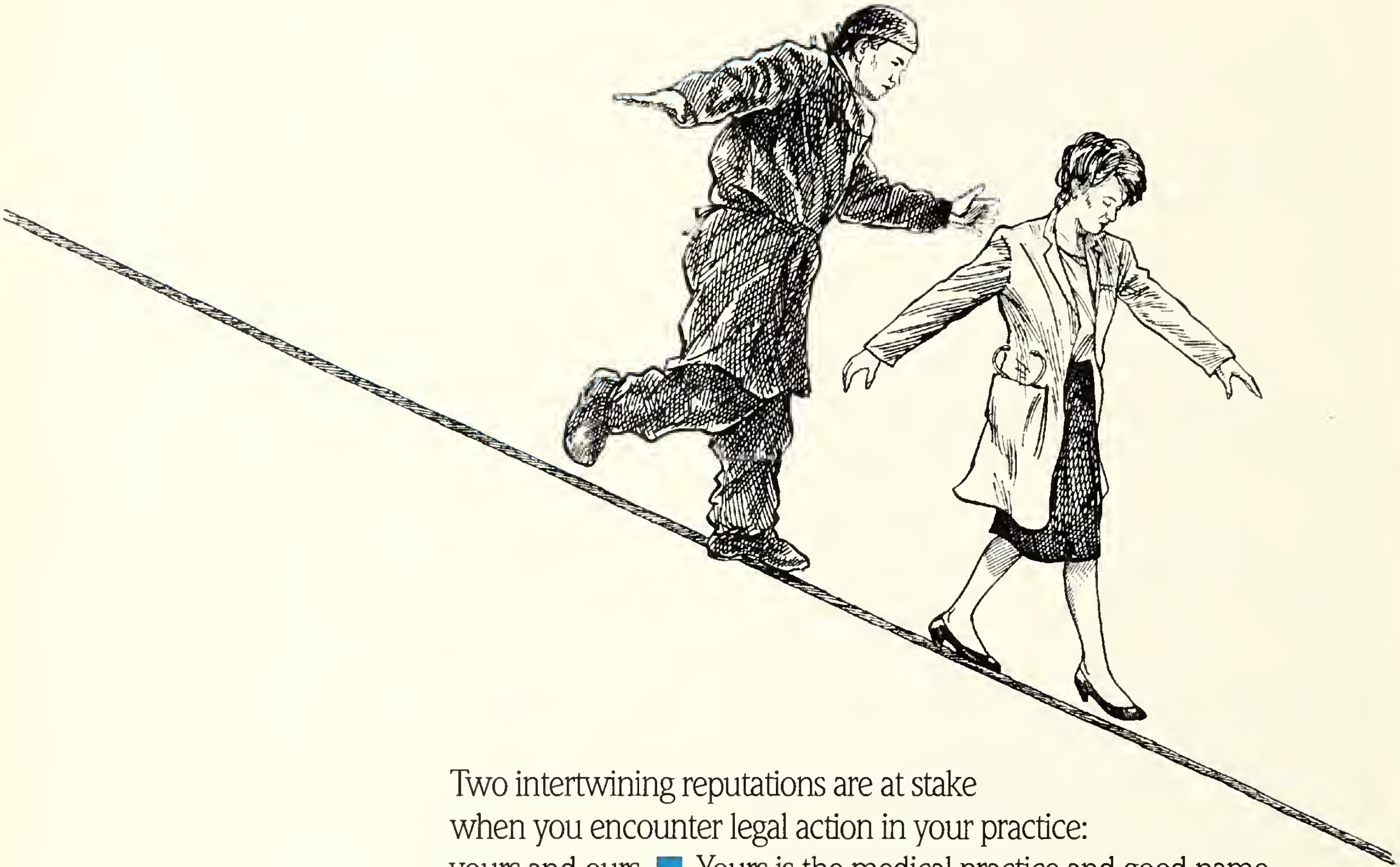
"Me" generation politics colors public policy, influencing decisions such as what rights are guaranteed persons under the Constitution, what happens when one person's rights infringe on the rights of another, and who is considered a competent person in our society. Issues of who has to be reckoned with, and when.

Society often gridlocks while these issues are clarified. Governments, courts, churches, corporations, and other entities, which function en masse if at all, stall because they may encroach upon the "natural" rights (alienable and inalienable) of one group of persons at the expense of another.

This makes medicine prone to controversy in our society, Cassell believes, because it inherently deals with the fundamental question of who calls the shots, the physician or the patient? A physician, armed with statistical probabilities of disease and standards of care to

Continued on page 281

When Your Reputation Is On The Line.



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observe, faces professional and personal dilemmas when confronted by a strong-willed, 41 year-old, pregnant patient who wants to experience "natural" childbirth at home, or by a terminal patient who is afraid of what might happen to him while his life is prolonged by technology that is beyond his control.

Patients feel entitled to make fundamental decisions about essential treatment. Physicians, well trained and legally responsible decision makers, have difficulty sharing their medical autonomy. Informed consent, advance directives, patient self-determination forms, and other such devices have been developed to encourage give and take communication and understanding between care givers and the patient.

Cassell, speaking at the March 1992 Dr. Morton Bender memorial lecture on medical ethics held at Barnes Hospital, called for the assimilation of a new goal in medicine to renew patients' trust in physicians ability to heal persons. This goal is philosophical - make medicine human-based; not disease-based. How adequately a doctor meets this goal can be measured by his or her ability to recognize and treat suffering.

This focus is proper, he contends, because suffering has the power to destroy a sick person - to rob him or her of identity and dignity - even while the person technically still is alive. A doctor who practices human-centered medicine recognizes the power of suffering and, with the patient's participation, concentrates on relieving its de-humanizing effects, even if such treatment may not prolong life. Thus assured, patients trust that doctors will attempt to heal them as a whole person, not just their diseased part, to the extent that is medically possible.

Cassell's prescription for care is threefold: treat the person, not the disease; maximize the patient's function, not length of life; and minimize the suffering of the patient and the family. Physician heal when they use their skill (and courage) to become close enough to the sick person to fathom their expectations and realistic capabilities. Healing care helps a patient to retain his or her "personhood" for as long as possible; technology that prolongs life but destroys the person compounds the suffering. Physicians who heal persons (rather than cure diseases) command respect in a society that values autonomy and freedom of choice.

Cassell acknowledges that many doctors intuitively use compassion and human- values with their patients. Many learn with experience that the scientific, disease-centered model ill-prepares them to deal effectively with humans - value-laden creatures who change through time and whose whole is greater than the sum of their parts.

Health will always be a precious and fragile commodity in an uncertain world. Researchers will find new diseases; different technology will be invented; resources will be more or less plentiful; and access will get better and worst. Governments and other entities will limit choices, manage care, or otherwise try to supervise the physician/patient relationship. Medicine, like life, always will be experienced in an imperfect world.

A focus on curing disease actually frustrates medicine, therefore, because its work never ends and it promotes a less sensitive approach to the patient's personhood. The outcomes of human-centered medicine can be as medically beneficial, and it is a goal that complements the physician and the patient's philosophical need to feel more human in a mass society.

Both the physician and the patient will be more at peace with their roles and obligations in this autonomous age. The cry of "Doctor, I want . . ." must be responded to on a humanistic level. ■

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The Use of Rectal Temperature to Monitor Heat Stroke

By Charles J. Ash, M.D., James R. Cook, M.D., Ted A. McMurry, M.D., and Cecil R. Auner, M.D.

The diagnosis and treatment of heat stroke has relied on rectal temperature monitoring. A twenty-year retrospective review of heat stroke and heat exhaustion victims reveals a 24% mortality and a 33% incidence of overcooling. An experimental protocol using hot and cool baths demonstrates marked rectal temperature lag during periods of rapid body heating and cooling. Auditory canal temperature measurements provide an attractive alternative to rectal temperature measurement during rapid temperature change. Rectal temperature monitoring of heat stroke victims is not recommended. This work suggests that shivering during treatment of heat stroke might be the result of vascular and brain overcooling with elevated rectal temperatures.

Heat stroke is a critical condition of high body temperature and central nervous system (CNS) symptoms caused by excessive heat exposure. It is associated with a high mortality.[1-3] Classical heatstroke is often seen in elderly patients with associated illnesses. During heat waves, the incidence of heat stroke can reach epidemic proportions.[4] Exertional heat stroke is seen more often in young, healthy individuals and is accompanied by more marked dehydration and enzymatic changes. This type may occur in epidemic fashion at sporting events or military exercises.

Heat exhaustion may precede exertional elevated body temperature. Rapid cooling of heat stroke victims is considered essential and rectal temperature monitoring is routine.[5] In most medical texts, ice water baths are recommended as the preferred treatment.[6]

In other areas of medicine the use of rectal temperature as a measure of body temperature during rapid temperature change has been questioned.[7-9] For example, in cardiac bypass surgery requiring blood cooling, rectal temperatures are not used to monitor body temperature because of a recognized lag in response at that site. Instead, direct blood temperature monitoring is used, usually in the pulmonary artery. In anesthesiology, a patient's body temperature is often monitored at the bladder, ear, forehead or esophagus,[10] to guard against malignant hyperthermia, another serious condition associated with high mortality. Brengelmann recently described the problems in his chapter "The Dilemma of Body Temperature Measurement" in the two volume work *Man in Stressful Environments. Thermal and Work Physiology.*[8] He feels that esophageal and oral temperatures are more accurate measures of changing internal body temperature.

The lack of a good measure of body temperature during rapid change has apparently caused a great deal of confusion in defining and classifying heat injury. Although core temperature and rectal temperature are not synonymous, the terms are often used interchangeably in the charts of heat stroke victims. Rectal temperature is still the method of measurement in physiology laboratories[11] as well as many centers that treat large numbers of heat stroke victims.[1] Much of the basic animal research on severe hyperthermia and tissue death has relied on rectal monitoring.[12]

The diagnosis and treatment of life-threatening thermal injuries become more problematic if rectal temperature measurement is not an accurate measure of internal brain and blood temperature.

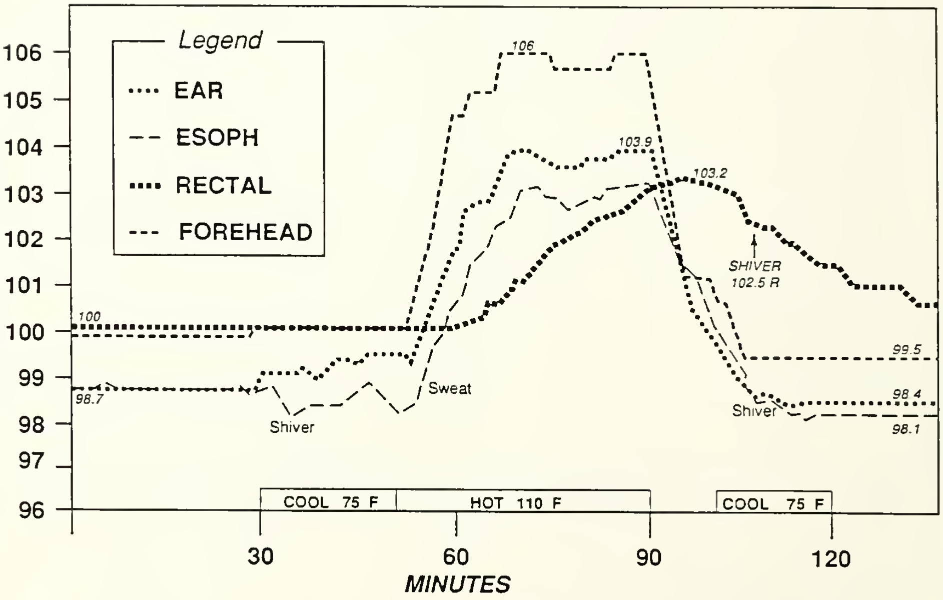
The temperature of the tympanic membrane gives an accurate representation of rapidly changing brain temperature. However, measurement of the tympanic membrane temperature with a direct contact probe is painful and risky.[13] Recently, an infrared thermopile radiometer was introduced and is now being used in emergency departments, recovery rooms and pediatric departments. The device is designed to measure the temperature of the tympanic membrane accurately without direct contact or discomfort. Its ability to measure temperatures over a wide range -- 60°F - 115°F (16°C - 46°C) -- is a decided advantage, and the fact that it uses disposable probes and avoids contact with mucous membranes makes it even more desirable. The literature is still quite sparse, and there have been mixed reports regarding the accuracy of this method.[14-17]

Methods and Materials

In order to determine the methods of temperature measurement and treatment, we reviewed the charts of all patients with a discharge diagnosis of heat stroke or heat exhaustion at St. John's Regional Health Center and Lester E. Cox Medical Center, Springfield, during the years 1970-1980. Charts were included only if there were CNS symptoms, history of temperature above 100°F (37.8°C) and environmental exposure without a final diagnosis of infectious disease. Particular attention was given to temperature monitoring in the immediate post-treatment period.

To demonstrate the rapidity and accuracy of temperature responses during rapid thermal transients, we compared various measurements of internal body temperature simultaneously, including esophageal (T_{ESOPH}) or oral (T_{ORAL}), rectal (T_{RECTAL}), auditory canal (T_{EAR}) and forehead (T_{SKIN}). A similar model demonstrating the rapid and comparable response of esophageal and oral measurement did not include (T_{EAR}) or (T_{SKIN}).[11]

Two rectangular whirlpool baths large enough to permit total immersion were utilized. One bath was filled with cool tap water. Water temperature T_{H2O} was 75°F (23.9°C). The other bath was filled with hot water and maintained at 107°F-110°F (41.7°C-43.3°C). The agitator was used to maximize heat transfer. A wet suit hood was used in the hot bath to reduce heat exchange from the head and neck by radiation and evaporation. The onset of shivering and sweating were recorded. T_{H2O} was monitored constantly. T_{ESOPH} was assumed to represent internal body temperature accurately and was monitored at a distance of 32 cm from the incisors with a Mon-A-Therm thermometer model 6500 and a YSE series 700 esophageal probe. T_{RECTAL} at a depth of 10 cm was measured with a YSI telethermometer and a series 400 rectal probe. T_{ORAL} was measured in the sublingual sulcus with an electronic clinical thermometer (Diatek 500). T_{EAR} was measured using an infrared thermopile radiometer (ITT: First TEMP, Intelligent Medical Systems, Inc., Carlsbad, CA), T_{SKIN} was measured with a liquid crystal tape applied to the forehead (Crystaline, SHARN Inc.). Temperatures were recorded every minute whenever possible. The subjects, CA (age 60) and TM (age 36), were in excellent health. Auditory canals were clear. Informed consents were obtained.



The initial subject (CA) was exposed to cool water for several minutes to demonstrate temperature changes at different sites as the result of shivering. Both subjects were then immersed in hot baths until internal temperatures had risen significantly. The first subject was allowed to cool partially before returning to the cool water to avoid too precipitous a drop of internal temperature. The second subject was younger and less hyperpyrexia was produced; he was transferred immediately to the cool water. Details of exposure methods appear later in this paper.

Reproducibility of T_{EAR} and T_{SKIN} was tested at resting conditions with varying ambient temperatures. The temperature of the auditory canal was verified using a clinical digital thermometer (BD) and a Diatek 500 adapted with a narrow surface probe that would fit inside the auditory canal. Forehead and auditory canal temperature measurements were recorded after exposure to cool outside air, room temperature, and an incandescent light source placed one inch from the forehead or ear.

Results

All cases fitting the criteria are included in Table 1. There were 21 patients and a 24% mortality. Poor prognosis was suggested by deep coma and high fever. Temperatures recorded as "core temperatures" had been measured rectally without exception. Seven patients (33%) demonstrated significant hypothermia in the recovery period. Only one patient was actually treated in an ice water bath, although several had ice bags and ice water applied locally. Cooling in transit in air conditioned ambulances appeared to be quite efficient when temperatures at the scene and later in the emergency room were compared. Seventy-one percent of patients were elderly and sedentary. Only one physician was involved in the treatment of more than one case. As can be seen, this is an uncommon condition in this locality and has not been seen in epidemic form.

Experimental Study

The results of the first study are recorded in Illustration 1. After fifteen minutes, CA was immersed to the neck in the cool bath. T_{H_2O} was 75°F (23.9°C). Esophageal temperature decreased slightly. After shivering for 15 minutes he was immersed in the hot bath (T_{H_2O} 110°F {43.3°C}) until T_{ESOPH} was maintained between 102°F (38.9°C) and 103°F (39.4°C) until T_{RECTAL} had nearly stabilized. The time of onset of sweating and shivering were recorded. Upon leaving the hot bath and being exposed to room air and fanning, all measurements of temperature dropped rapidly except T_{RECTAL}. The forehead strip registered significantly higher than other locations. Its response to rapid cooling and heating was brisk but exaggerated. Thirty minutes after shivering had begun, T_{RECTAL} had still not returned to normal.

Shivering did not occur when the oral or esophageal temperature were elevated, but clearly did occur with an elevated rectal temperature - 102.5°F (39.2°C). Esophageal temperature drop was rapid and marked, although the water temperature was never below 75°F (22°C). Water directly from the tap was cool enough to produce shivering. Body temperatures were consistently lower than baseline temperatures after hot bath exposure. This confirms our previous results[10] and seems to indicate a lowered shivering setpoint after hot bath exposure.

Results of the second study (subject TM) are recorded in Illustration 2. Sweating occurred three minutes after entering the hot bath. Marked rectal temperature lag was again noted. Response of the forehead strip was again exaggerated, but oral, ear and forehead temperatures responded quite rapidly. Shivering occurred eight minutes after transfer to the cold bath, and a slight sensation of faintness occurred. Rectal temperature lag was obvious, remaining at 101°F (38.3°C) when shivering occurred. All other measurements had dropped below baseline values. The shivering setpoint was again depressed. The pulse became quite slow (44 beats/min) after exposure to cool water. Recovery was uneventful as the bath water was warmed to comfort levels.

Results of changes due to ambient temperature are summarized in Table 2. The liquid crystal plastic forehead skin strip responded in an exaggerated fashion to temperature elevations and was markedly affected by ambient temperature.

ILLUSTRATION 2

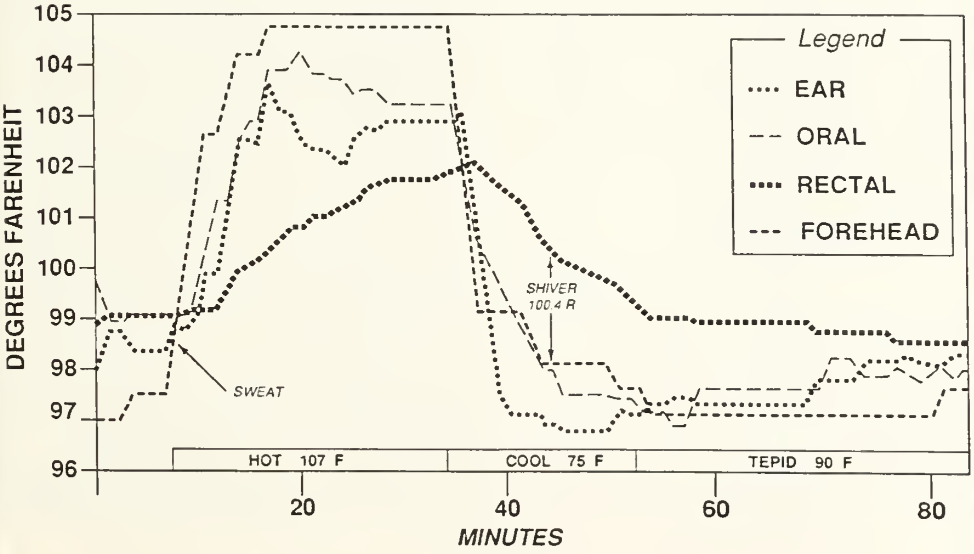


Table 1

AGE	TEMP °F	ONSET	CNS	TREATMENT	HYPOTHERMIA	RESULT	REMARKS
(F)76	106°	Collapsed	Coma	Ice bath to 102°F	No	Recovered	Aphasic- Steatorrhea
(m)76	106.4°	Collapsed Burning Trash	Coma Seizures	Ice Bags ER	97.2°F	Recovered 2 Weeks	High Enzymes - Blisters on Legs, Awakened, Ice Bag - Nursing Home, Confusion.
(M)78	105°	Collapsed House Trailer	Coma	Sponged Cooling Blanket	No	Recovered 2 Days	Temp Norm in Six Hours
(M)24	108°	Collapsed Roofing	Coma Seizures	Ice LMD Ambulance Air Conditioning	96.0°F	Recovered 4 Days	Vomited - CPK 1200, Shivering, Rigid in ER (104°F) LDH 309, SGOT 111, Sugar 341
(M)73	106°	Collapsed	Coma	Ice Packs	95.5°F	Died 2 Days	Shock- Chronic Lung
(M)38	103.8°	Collapsed Race	Coma	Ice Bags and Fan 5 Hrs. to 100.5°F	Probable	Recovered 5 days	Shivering 45 Min.,Shaking 3 Hrs., Elevated Enzymes, Amnesic Vomited
(F)76	103°	Collapsed Hot House	Coma	Air Conditioner and Wet Sheets - Awoke in Ambulance	96.4°F	Recovered 4 Days	Uncomplicated Recovery, Demented - Prev. Lupus
(F)92	108°	Collapsed Invalid	Coma	Sponged	No	Died Same Day	NA 128, PO2 56, K 3, PCO2 20, Sug 484, PH 7.46
(M)76	109.6°	Collapsed in Parked Car	Coma Seizure	Iced Down - IV Fluids - Cooling Blanket - IV Valium - IV Steroids	No	Recovered 3 Days	PLAT 190,000 ,PRO Time 15, LDH 283, SGOT 123, PH 7.43, PCO2 30, Sug 132 Purpura
(M)79	108°	Collapsed on Street	Coma Shock Dry	Cooling Blanket IV Fluids		Died Same Day	
(F)68	106.1°	Collapsed Hot House	Coma Shock	Ice in Ambulance IV Fluids	No	Recovered 9 days	PLAT 88,000, LDH 40, SGOT 16, LDH 1160, Bili 1.6, PCO2 30, CPK 6160
(M)79	102.8°	Wall Papering	Confused	Non Agressive	No	Recovered 3 days	Uneventful
(M)18	101.9° 107°	Collapsed Yard	Confused Coma	IV's 4 Hrs Cooling Blanket	95. 4°F	Died 1 day	Vomit - Diarrhea - Sugar 1700, High Enzymes
(M)14	103.6°	Spectator Ball Game Hemiplegic	Lethargic	Sponged At Home	No	Recovered 4 days	
(M)75	108°	Collapsed Hot Hotel	Coma	CPR - Cooling Blanket	97°F	Died 3 days	Seizure - Vomited, Autopsy
(M)32	104.4°	Welding	Ataxic	Cold Water LMD Cold Towels ER	No	Recovered 1 day	Uneventful
(F)74	104.3°	Collapsed Hot House	Lethargic	Air Conditioner in ER	No	Recovered	Uneventful, 4 days
(F)70	107.6°	Collapsed with fans	Coma Seizure	Ambulance Air Cooling 30 Min	No	Recovered 5 days	Electrolytes Normal, Sug 600
(F)70	106°	Collapsed Hot House	Semi Coma	Cold Towels Ice Gastric Lavage Fans	No	Recovered 3 days	Sug 327, LK 164, SGOT 14
(F)73	105°	Collapsed Hot House Polio Cachetic	Lethargic Confused Dry	Ambulance 20 Mile IV Fluids	No	Recovered 3 days No Cooling in Hospital	
(M)59	107°	Collapsed Walking Mental Nevada	Coma Seizure Shock	Ice Packs Haldol Fan 60 Min	No	Recovered 17 days	Ph 7.5, PCO2 32, Neurological Work-Up Combative After Treatment

Discussion

The occurrence of post-treatment hypothermia in 33% of cases suggests the patients were overcooled due to the lag in rectal temperature change. The experimental demonstration suggests shivering during treatment of heat stroke might indicate below normal brain temperatures, even though rectal temperatures are elevated.

The FirstTemp radiometer was accurate and responsive to sudden changes in the internal temperature. However, it actually measures the temperature of the auditory canal, not the tympanic membrane, and is affected somewhat by ambient temperature (Table II). The "cal" setting corresponds to the surface temperature of the auditory canal. To obtain an "oral," "rectal" or "core" reading, a factor of 0.7°F (0.4°C), 1.4°F (0.8°C) or 1.7°F (0.9°C) is added to the surface or "cal" temperature. Despite this arbitrary "marketing" innovation, the radiometer appears to be a satisfactory alternative to oral measurements in the comatose patient, and far superior to rectal measurements during rapid body temperature changes.

Tap water below 60°F (15.6°C) is very cold. In 1973 Shapiro developed an excellent dog model and demonstrated the direct relationship of degree of temperature elevation and mortality. He described pathological changes identical to those found in humans.[17] He and Magazanik used this same model in 1980 to monitor heat stroke treatment with water baths of various temperatures and concluded that ice water offered no more rapid cooling than tap water at a temperature of 60°F (16°C).[18] The only two dogs that died during treatment were exposed to ice water. In common with all other heat stroke studies we reviewed, they used rectal temperature to monitor changes. Some of the dogs awakened and tried to escape the cold water bath well before rectal temperature returned to normal. Panting also stopped while rectal temperature was still elevated. Rectal temperature lag might well explain these findings. Other articles have suggested alternative cooling methods including hot water sprays[1] and helicopter fanning.[19]

Table II

AMBIENT TEMPERATURE	FOREHEAD TEMPERATURE	EAR TEMPERATURE	ORAL TEMPERATURE
OUTDOORS 41°F	88°F	96.7°F	98.8°F
HOT LIGHT 100°F	106°F	99.2°F	98.8°F
INDOORS 70°F	97°F	98.7°F	98.8°F

Conclusions

A patient's response to treatment of heat stroke should not be monitored by rectal temperature measurement. The rectal temperature lags well behind vascular and brain temperature during rapid temperature change.

Measurement of auditory canal temperature appears to provide an attractive alternative when oral and esophageal temperature measurements are not practical.

Measurement of forehead skin temperature with liquid crystal strips appears to be very inaccurate and is markedly influenced by ambient temperatures.

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ACKNOWLEDGMENT: Gail McDaniel, art director at Lester E. Cox Medical Center, created the graphs for this article.

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The Role of Antibiotics in the Management of Acute Exacerbation of Chronic Bronchitis *(Answers to Some Commonly Asked Questions)*

By Beata A. Skudlarska and Graham C. Scott, M.D.

More than 7 million Americans suffer from chronic bronchitis,[1] and it has been estimated that each of these patients will experience between one and four acute exacerbations a year.[2] This problem constitutes a major social and financial burden on our society. Despite the prevalence of this disorder there is still considerable disagreement among physicians as to appropriate treatment, especially with regard to the use of antibiotics. Though the role of antibiotics is unclear, the pharmaceutical industry is trying to obtain regulatory approval of several new antibiotics for use in exacerbations of chronic bronchitis. We examine the role of antibiotics in the management of chronic bronchitis.

Discussion

Question: What is chronic bronchitis?

Answer: Chronic bronchitis refers to ". . . a condition of chronic or recurrent excess mucus secretion into the bronchial tree . . ."[3]. To qualify as chronic, the condition should ". . . occur on most days, for at least three months of the year, for at least two successive years . . .".

Excess mucus secretion is empirically defined as the production of any sputum, whether expectorated or swallowed, and in most instances is accompanied by chronic cough. Although not explicitly stated in this definition, it is generally held that excess mucus production is an important cause of airflow obstruction[3], and chronic bronchitis implies expiratory airflow limitation. In chronic bronchitis, this excess secretion of mucus is not due to other diseases, such as bronchiectasis or tuberculosis.

The term "Chronic Obstructive Pulmonary Disease" (COPD) does not refer to one particular entity. This term describes several disorders characterized by marked airflow obstruction, of which chronic bronchitis is one. The major features of this disorder are shown in Table 1.

Question: Why are patients with chronic bronchitis prone to developing respiratory tract infections?

Answer: The pathogenesis of an acute exacerbation in chronic bronchitis is still not fully understood. The major predisposing factors in patients with chronic bronchitis are listed in Table 2. Bacterial infections are an important cause, but factors such as viral infections of the respiratory tract, allergic reactions, and increased exposure to cigarette smoke may account for more than half of all cases.[4,5]

Question: What are the most prominent signs and symptoms of an acute exacerbation of chronic bronchitis?

Answer: The most prominent features of an acute exacerbation of chronic bronchitis are shown in Table 3. Physical examination is usually noncontributory, but may reveal an increase in crackles and wheezes. A chest roentgenogram can help distinguish an exacerbation

TABLE 1

The syndrome of Chronic Obstructive Pulmonary Disease (COPD) consists of four major components.

A. **Chronic Mucus Hypersecretion** (a clinical diagnosis based on history),
B. **Pulmonary Emphysema** (destruction of parenchyma),
C. **Airways Hyperreactivity** (asthma/reversible airflow limitation),
D. **Changes in Small Airways** (including bronchiolitis).

Each major component may exist alone but usually they exist concomitantly.

from pneumonia, but often is unremarkable. The white blood cell count and erythrocyte sedimentation rate may be elevated, but are often within normal limits.

Question: Is a Gram stain and culture of expectorated sputum of any value in patients with an acute exacerbation of chronic bronchitis?

Answer: As yet no consensus has been reached as to the value of either a Gram stain or culture of sputum in these patients. An excess of neutrophils on a sputum Gram stain is believed to indicate a bacterial infection, but may also be present in viral infections, or even in noninfectious processes.[5,6] Certainly the presence of more than 25 white blood cells, and fewer than 10 epithelial cells per low power field is presumptive evidence of a bacterial respiratory tract infection.[6]

The number of bacteria present on a Gram-stained sputum sample may help distinguish an acute bacterial infection from "stable" bronchitis. Baigelman[7] et al examined over 1,000 Gram-stained sputum samples and concluded that the presence of 18 or more organisms suggestive of *Hemophilus influenzae*, or eight or more of *Streptococcus pneumoniae* per immersion power field is strongly indicative of a bacterial origin for the acute exacerbation. They also suggested that the presence of predominantly Gram-negative bacilli, which are not normally seen, is also suggestive of an acute bacterial infection.

The value of obtaining a sputum culture in most cases of exacerbation of chronic bronchitis is unclear. Analysis of transtracheal aspirations have shown that the airways of patients with chronic bronchitis are often colonized with *Hemophilus influenzae* or *Streptococcus pneumoniae*.[8,9] Other investigators have argued that the tracheobronchial tree is usually sterile,[10] and that sputum cultures can assist the physician in the diagnosis and management of acute exacerbations. We do not recommend obtaining cultures routinely.

Paterson et al showed there was no difference in the resolution of symptoms between those patients in whom the reports of sputum cultures were regularly reviewed and used to determine management, and those in whom such reports were not reviewed[11].

In a recent study at our institution, we analyzed sputum Gram stains in 115 consecutive patients who had symptoms of an acute exacerbation of chronic bronchitis (increased dyspnea and cough, and a change in the amount and appearance of sputum). A sputum Gram stain with less than 10 epithelial cells and more than 25 polymorphonuclear cells per low-power field was seen on 95 occasions (83%). All of these patients were treated with an oral antibiotic agent. However, we did not find any difference in the rate of improvement or resolution of symptoms between those with a "negative" or "positive" sputum culture.

Another factor that has not received much consideration is the experience of the physician reading the Gram stain. Preliminary data from an ongoing study suggest that internists who are not working in the field of infectious diseases are not consistent in their interpretation of Gram stains.[12]

The final decision about Gram staining a patient's sputum and sending it for culture rests with the physician, and should be based upon the history and physical examination. We advocate obtaining and reading a Gram-stain sputum before instituting antibiotic therapy in the patient, but see little value in routinely obtaining a sputum culture (see next question).

Question: Which bacterium is likely to be responsible for an acute exacerbation of chronic bronchitis?

Answer: Organisms believed to play a major role in bacterial exacerbations of chronic bronchitis are *Hemophilus influenzae, Streptococcus pneumoniae,* and *Branhamella catarrhalis* (now renamed *Moraxella catarrhalis*).[13-15] Bacteria, such as *Staphylococcus aureus, Pseudomonas aeruginosa,* and other Gram-negative bacilli are only cultured in

TABLE 2

Major Predisposing Elements to the Development of Acute Exacerbations in Chronic Bronchitis

A. **Excess of Mucus Secreting Goblet Cells**
   Normally these account for 25% of all epithelial cells of the major bronchi, but in chronic bronchitis may increase dramatically.

B. **Hypertrophy of the Submucosal Glands**
   This may result in the production of a very thick mucus that is difficult to expectorate.

C. **Loss of Normal Ciliated Epithelium**
   Impairment of the normal mucociliary elevator which makes removal of inhaled bacteria difficult and allows their persistence and growth in the airways. These bacteria provide a reservoir for an acute exacerbation whenever host defense mechanisms are compromised.

D. **Defects in the Local Host Immune System**[33,34]

TABLE 3

Signs and Symptoms of an Acute Exacerbation of Chronic Bronchitis

A. Increased cough.
B. Increased production of mucopurulent sputum.
C. Increased dyspnea.
D. Fatigue, chills, pleuritic pain and increased wheezing (less common).
E. Fever, lethargy, and general malaise (suggestive of a more serious problem such as pneumonia).
F. Cyanosis.
G. Orthopnea and chest pain (in patients with left ventricular failure)[10].

about 5% of cases.[16]

In the study mentioned above, we cultured expectorated sputum from 83 patients with a clinical diagnosis of an acute exacerbation of chronic bronchitis, and in whom a Gram stain showed more than 25 polymorphonuclear cells and less than 10 epithelial cells per low-power field. We specifically identified each organism that was cultured, even though routinely a large number would have been reported as normal mouth flora.

These patients were divided into three subgroups:
(I)Those who experienced one exacerbation, and in whom only one causative organism was cultured [53 patients (55.8% of all cases)]
(II)Those who experienced one exacerbation, in whom more than one organism was cultured (16 patients, 40 different bacteria)
(III)Those with recurrent exacerbations (10 patients; 22 episodes; 25 bacteria).

In four patients, no organism was cultured.

In the majority of cases a single organism was cultured (see Table 4). When a single organism is obtained, and that organism is known to be a common pathogen, it is a logical step to believe this agent to be the cause of the acute exacerbation. However, in a large number of patients, organisms that are not considered pathogens, such as *alpha Streptococcus* and other *Streptococcus* species were cultured. The significance of these findings is unclear. As we have discussed before, the value of sputum cultures appears to be limited.

*Question:*Is there any benefit from using antibiotics in the treatment of acute exacerbations of chronic bronchitis?

Answer: Numerous studies have attempted to determine the value of antibiotics in the treatment of patients with acute exacerbations of chronic bronchitis.[2,17-25] However, they are sometimes difficult to interpret because of such factors as variable selection criteria, lengths of therapy, lack of controls, different definitions of improvement, and sometimes questionable statistical analysis.

Older studies failed to show any statistically significant benefit from the use of antibiotics,[21,22] whereas more recent studies have suggested that they are of value.[2] Anthonisen[2] observed 173 patients with chronic bronchitis for 3.5 years, and noted 362 exacerbations. In a double-blinded, randomized fashion patients were placed on either a placebo, or one of three antibiotic regimens: amoxicillin 250 mg four times a day, trimethoprim/sulfamethoxazole 160 mg/800 mg twice a day, and doxycycline with initial dose 200 mg daily followed by 100 mg daily. Each patient was graded according to the severity of their symptoms.

Resolution of symptoms after 21 days was reported in 55% of the placebo group and 68% of the antibiotic

TABLE 4

*Organisms Obtained From Sputum Cultures in 95 Consecutive Patients With an Acute Exacerbation of Chronic Bronchitis**

Organism Cultured	N	% of total group
Group I. (53 patients)		
Hemophilus influenzae,	7	
Branhamella catarrhalis,	3	24.5
Streptococcus pneumoniae.	3	
Staphylococcus aureus,	3	
Escherichia coli,	1	10.0
Pseudomonas aeruginosa.	1	
Alpha streptococcus	17	
Streptococcus species.	5	
Klebsiella pneumoniae	1	
Serratia marcescens	1	65.5
Enterobacter cloaceae	2	
pseudomonas species	4	
acinetobacter calcoaceticus	3	
Neisseria meningitidis	1	
Enterobacter aerogenous	1	
Group II. (16 patients, 40 bacteria)		
Hemophilus influenzae,	4	
Branhamella catarrhalis,	3	25.0
Streptococcus pneumoniae.	3	
Staphylococcus aureus,	3	
Escherichia coli,	2	17.5
Pseudomonas aeruginosa.	2	
Alpha streptococcus	3	
streptococcus species	0	
Klebsiella pneumoniae	6	
Serratia marcescens	2	57.5
Enterobacter cloaceae	2	
pseudomonas species	3	
Aromonas hydorphila	2	
Klebsiella oxytocia	2	
Neisseria mucosa	1	
Serratia feciens	1	
Hemophilus parahemoliticus	1	
Group III. (10 patients, 22 episodes, 25 organisms)		
Hemophilus influenzae,	1	
Branhamella catarrhalis,	5	32.0
Streptococcus pneumoniae.	2	
Staphylococcus aureus,	4	
Escherichia coli,	3	36.0
Pseudomonas aeruginosa.	2	
Alpha streptococcus	4	
streptococcus species.	1	20.0
Pseudomonas fluoersences	1	
Hemophilus parahemoliticus	1	12.0

*See text for definition of each group.

group respectively. This was a statistically significant difference. The overall failure rate was significantly greater in the placebo group (42%), than in the antibiotic group (29%). A placebo proved to be as effective as an antibiotic in the treatment of those in the "mild" category, but became less so as the severity increased, reaching a 53% failure rate in the "severe" category.

Despite discouraging study results, the use of antibiotics is still "in fashion." We recommend their use in severe and rapidly progressing exacerbations of chronic bronchitis, especially if the Gram stain of the sputum suggests a bacterial cause. In less severe cases, however, the fact that 50% of exacerbations "respond" to placebo cannot be overlooked.

Question: Which antibiotic should be used when the decision to treat an exacerbation of chronic bronchitis has been made?

Answer: The results of previous studies are unclear as to the appropriate antibiotic to use.[4,5] Chodosh[5] reported that despite reservations by others, ampicillin, bacampicillin, amoxicillin, doxycycline, minocyclin and trimethoprim/sulfamethoxazole have a low treatment failure rate and a relatively long infection-free period. In a recent study comparing ampicillin with ciprofloxacin, both drugs were shown to have similar efficacy, but ciprofloxacin appeared to clear bacteria from the sputum more effectively[26].

In the above mentioned study we noted a high degree of ampicillin-resistant strains. In 43% of all cultures obtained, the organism was resistant to ampicillin. This contrasts with ciprofloxacin, to which fewer than 5% of strains were resistant.

The use of erythromycin, azithromycin, cefaclor and cephalexin have been associated with a high failure rate,[5,27] suggesting that these are not agents of "first choice." Some of the new antibiotics, such as ciprofloxacin (ß-lactamase resistant) or amoxicillin/clavulanate potassium (ß-lactamase inhibitor), have been shown to be superior in infections caused by beta-lactamase producing strains of *Branhamella catarrhalis* and *Hemophilus influenzae.*[28] However, a high incidence of recurrent infections with these regimens has been reported.[28-32]

Although there is great concern about the rising number of infections caused by strains resistant to "first choice" agents, the newer antibiotics still need to be evaluated before they are routinely used for the management of exacerbations of chronic bronchitis.

No single antibiotic can be recommended, but ampicillin, amoxicillin, doxycycline and trimethoprim/sulfamethoxazole are still considered "first choice" agents. Duration of therapy should be 10-14 days. If the case is complicated by pneumonia, subsequent relapse, or if a resistant organism is suspected in patients with multiple episodes, newer agents such as quinolones, second or third generation cephalosporins or agents with a beta-lactamase inhibitor should be used.

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Intranasal Melanoma

By Walter A. Schroeder, Jr., D.O., M.D.

Malignant melanoma is most commonly associated with cutaneous malignancies of sun-exposed skin, but melanoma also occurs in the mucosal surfaces of the head and neck. The symptoms of intranasal melanoma are most often associated with benign conditions that patients often ignore during the early course of the disease. Benign symptoms that persist require thorough evaluation to exclude neoplastic disease in which timely diagnosis and early treatment offers the best prognosis.

Approximately 20% of malignent melanomas occur in the head and neck.[1] Primary malignant melanomas of the nasal mucosa are unusual, with an incidence of 0.5% to 2.7%.[2,3]

Nasal obstruction and epistaxis are the two most common presenting symptoms. Patients may not seek medical attention, however, because these symptoms are nonspecific and believed to be self-limiting. Pain and swelling of the face and nose are usually late symptoms that may reflect advanced disease within the nasal cavity.

Most patients diagnosed with intranasal melanoma are in the fifth through eighth decade of life.[4] Though earlier reports noted a slightly greater incidence in women, reports published since 1960 show no definite gender predilection.[4]

Case Report

A 51-year-old white woman with a long-standing history of sinus symptoms was referred by her primary care physician for evaluation of nasal obstruction and failure to respond to decongestant and antibiotic therapy. She had received a diagnosis of Addison's disease several years earlier. The patient was also noted to have mild hypertension, which was well controlled medically.

At physical examination, a polypoid growth was found involving the right inferior turbinate. The growth was a flesh-colored mass that was firm and not tender upon palpation. The mass did not appear to be friable.

The patient's chest x-ray film revealed no active cardiopulmonary disease. Her blood chemistry profile was unremarkable.

A CT scan performed of the head and neck revealed a thickening within the right maxillary antral mucosa (Fig 1). There was no evidence of any bone destruction, nor any disease in the left nasal cavity or paranasal sinuses.

The patient underwent surgical excision of the intranasal mass via a lateral rhinotomy approach (Fig 2). The mass appeared to extend posteriorly to encompass the entire inferior turbinate. There was no extension into the maxillary sinus, though the natural sinus ostium was obstructed by inflammatory mucosa. The nasal septum itself was not involved. The lateral rhinotomy approach allowed visualization of both inferior and middle turbinates and provided adequate exposure for total excision of the lesion.

The nasal cavity was packed with petroleum-impregnated gauze after closure of the incision.

The pathology report of the permanent section revealed a primary malignant melanoma of the inferior turbinate (Fig 3,4).

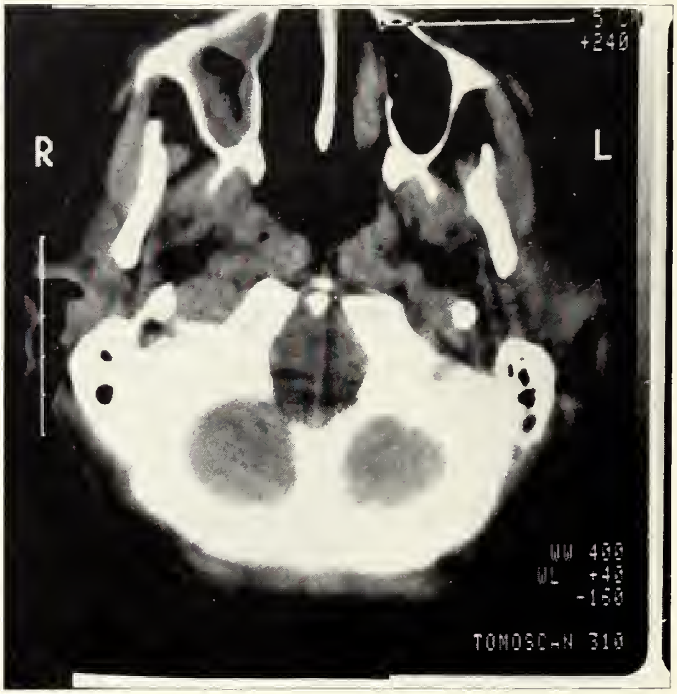

Figure 1
CT scan revealing musocal thickening of the right maxillary antrum.

Subsequent examination following surgery revealed no evidence of metastatic disease. The patient's funduscopic examination was unremarkable. The liver-spleen Tc-99M scan revealed hepatomegaly and findings that indicated mild hepatic parenchymal disease. No focal space-occupying lesion was identified. The patient was evaluated in consultation by a medical oncologist. No chemotherapy was recommended.

The patient underwent planned post-operative radiation therapy consisting of 3,600 rads during nine treatments. She remains free of her disease 18 months following surgery.

Discussion

Although Carswell used the term melanoma in 1835,[5] Lucke in 1869 described a surgical procedure for "a melanocytic sarcoma which arose from the nasal mucous membrane."[6] The first report in the American literature was described by Lincoln in 1885.[7] A report on the single largest series of mucosal melanoma was published by Ravid and Esteves in 1960.[4]

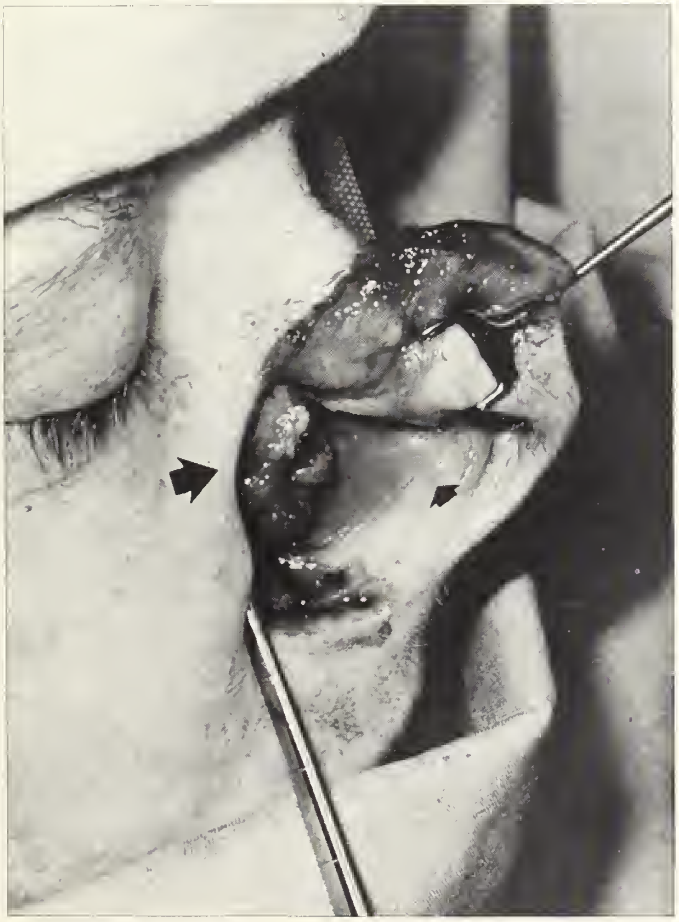

Figure 2
Intraoperative view of a lateral rhinotomy incision providing exposure of the inferior turbinate (large arrow) and view of the nasal septum (small arrow).

Trauma and chronic infection have been reported to be causative factors.[8,9] Many authors have reported the presence of polyps or a history of nasal polyposis in patients with mucosal melanoma.[1,4,10,12] Ravid and Esteves[4] reported nasal polyps had been removed one or several times "in a rather large number of patients." Five of 39 patients reported by Holdcraft and Gallagher[10] have a history of nasal polypectomy. Freedman et al[1] noted that nine of 56 cases of mucosal melanoma were diagnosed during routine nasal polypectomies. Snow et al[13] reported two of 11 patients had histories of repeated nasal

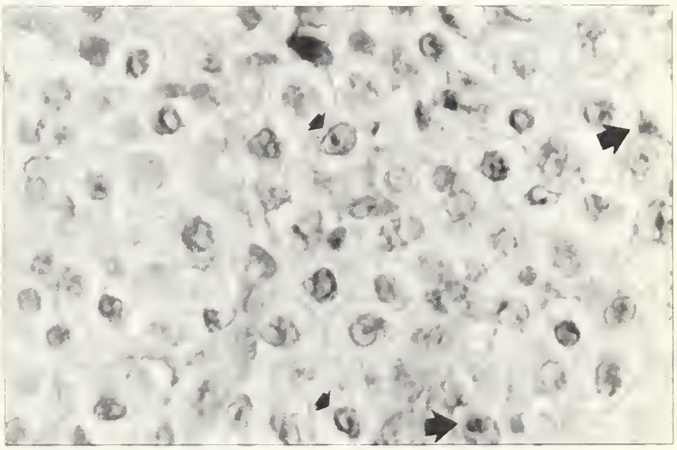

Figure 3
H&E stain. Melanoma demonstrating prominent pleomorphism of cells with vesicular nuclei and several cells showing macronuclei (small arrows). Focal darkened areas are melanin pigment (large arrows).

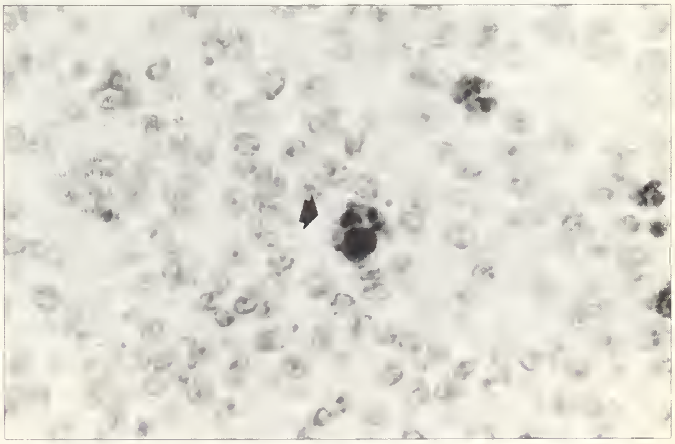

Figure 4
Fontana silver stain demonstrating melanin deposits.

polypectomies.

The gross appearance of intranasal melanoma varies from a friable polypoid or sessile mass ranging from pink to black in color. The friability explains why epistaxis is often a common symptom.[3,4,10] Melanin pigment is usually present in the primary tumor, but 10% to 30% of the tumors will be amelanotic.[3] The Fontana silver stain allows the melanin pigment to be more clearly observed (Fig 4).

Intranasal melanomas tend to show greater anaplasia and pleomorphism, often resembling a spindle cell sarcoma rather than a carcinoma.[4] This spindle cell pattern is more common in mucosal melanoma than in cutaneous melanomas.

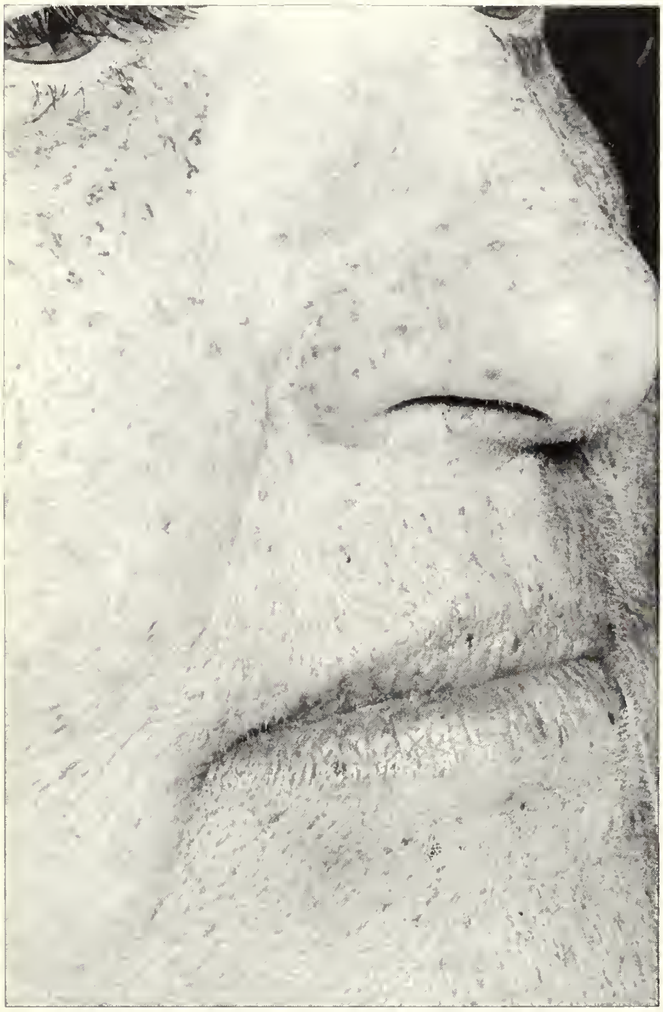

Figure 5
Lateral rhinotomy incision approximately one year following surgery. The incision remains well-camouflaged by the natural anatomical junction between the nose and cheek.

Depth of invasion is difficult to establish in mucosal melanoma because there are no histological landmarks analogous to the papillary and reticular dermis. Measuring the level of invasion, therefore, does not readily apply in mucosal melanoma.

Mucosal melanoma differs from cutaneous melanoma in a variety of ways. The size of a primary cutaneous melanoma relates to prognosis, but this relationship is not readily observed in mucosal melanoma.[11] Satellitosis is rarely observed in mucosal melanoma, but often seen in cutaneous melanoma.[11,13] Cutaneous melanoma more often have regional nodal metastases, but this is less commonly seen in mucosal melanomas.[1,11,14] Only 8% to 18% of patients with intranasal melanoma have cervical metastatic disease.[3]

Mucosal melanoma, as with cutaneous melanoma, remains a surgical disease.[1,3,13,15] Great emphasis is placed on complete excision of the melanoma. Radiation may be effective in combination with surgery.[16] Radiation is often used when surgical margins are positive, when complete surgical excision is not possible, or when combined therapy is desired. Radiation has also been used for local recurrence or when patients refuse surgery. Harwood's[17] review of 25 patients with mucosal melanoma who were treated with primary radiation therapy revealed complete local remission in eleven patients during a follow-up period of nine to 54 months.[15,17] Patients treated with a high dose fraction had better success rates.

Though chemotherapy may have value in the management of metastatic or recurrent disease, its role as primary treatment modality for mucosal melanoma is not established.[15]

The prognosis for patients with mucosal melanoma remains poor. Approximately 55% to 65% of patients with mucosal melanoma of the nose or paranasal sinuses will have either local recurrence or metastatic disease within the first year of treatment. Most common is local recurrence; distant metastatic disease is most commonly found in the lungs, lymph nodes or brain.

Conclusion

The persistence of common nasal symptoms usually associated with benign conditions may represent malignancy that will best respond to timely diagnosis and treatment. The presence of any intranasal mass warrants attention, and malignancy must be included in the differential diagnosis.

The treatment of mucosal melanoma, as with cutaneous melanoma or melanoma at other anatomical sites, is surgical excision. Radiation as adjunctive therapy may be included in the treatment regimen.

Though usually associated with sun-exposed skin, melanoma can occur in the mucosal surfaces of the head and neck. Pigmented lesions of the mucosal membranes should be promptly evaluated. This case demonstrates the common clinical symptoms, the use of the Fontana stain to assist in pathological diagnosis, and the role of the lateral rhinotomy approach for adequate exposure and surgical excision of this unusual disease.

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Malignant Tumor of the Colon Metastatic to the Epididymis As a First Sign of Recurrence of Colon Cancer

By Raul O. Parra, M.D., John Boullier, M.D. and Donald J. Mehan, M.D.
St. Louis, Missouri

Metastatic tumors to the epididymis are rare. Most reported cases represent another occurrence of widespread disease. This is a report and review of the literature of a case of adenocarcinoma of the colon three years post resection in which the only site of recurrence was in the epididymis.

The clinical finding of an enlarged and indurated epididymis is not uncommon to urologists, however, a metastatic tumor to this organ is not usually included in the initial differential diagnosis. We present what we believe to be the fourth case in the literature of colon cancer metastatic to the epididymis and the first such case in which the epididymis is the only site of metastasis from an adenocarcinoma of the colon.

Case Report

An 87-year-old white man had a one-month history of left-sided scrotal pain. He also complained of having night sweats and an increase in the size of his scrotum. He denied having dysuria, urinary urgency or hematuria. He also denied having weight loss or fatigue. He had had adenocarcinoma of the colon treated with sigmoid resection and primary anastomosis three years previously. Pathological findings at that time indicated he had a Duke's Stage B, moderately differentiated adenocarcinoma with extension into the serosa. Eight of the lymph nodes excised were negative for metastatic disease.

At initial physical examination, the patient was noted to have a large hydrocele on the left with a hard nodular cord structure which was thought to be epididymis. The testicle and epididymis were not readily identified by physical examination. A scrotal ultrasound revealed a large hydrocele and an irregular mass consistent with the epididymis.

Laboratory Data

The patient's white blood count was 7.6, hemoglobin and hematocrit 14 and 43, respectively. Urinalysis showed 0-2 white blood cells per high powered field, no bacteria and no hematuria. A urine culture was also obtained, which after 48 hours showed no growth. His serum creatinine was 1.4, his liver function tests were within normal limits and his prostatic specific antigen and prostatic acid phosphates, 0.5 and 0.2 ng/dL, respectively. He was scheduled for an inguinal exploration and possible orchiectomy.

At operation a very large epididymal mass was found intimately adhering to the cord structures. The specimen was removed in toto through a left inguinal approach. Pathological analysis revealed a specimen which weighed about 100 g, measured 3.5 x 3.5 x 2.5 cm, and was soft and tan in color. This tumor was infiltrating the soft tissues around the vas deferens and had invaded and replaced most of the epididymis. Microscopic examination revealed adenocarcinoma of moderate differentiation consistent with colon cancer (Fig 1) and (Fig 2). Areas of normal epididymis were noted adjacent to the mass. The testicle consisted of atrophic tubules without evidence of spermatogenic activity.

Postoperatively, the patient underwent a flexible sigmoidoscopy procedure that did not reveal a recurrence of cancer at the original surgical site. He then underwent colonoscopy, which gave unremarkable findings except for pseudomembranous colitis. A bone scan was found to be negative for metastatic disease. His serum carcinoembryonic antigen level (CEA) was within normal limits (1.7) as were his liver function test results. A CT scan of the abdomen and pelvis was negative for any signs of recurrent disease. To this point, the patient remains well with no evidence of recurrence.

Discussion

Cancer of the epididymis is rare. Primary tumors are themselves not common and metastatic disease to the epididymis is even more uncommon. Only five cases of metastatic disease to the epididymis are found in the literature prior to 1960.[1] Broth found that in 278 cases of tumor of the epididymis, 13 (5%) were metastatic in origin.[2] A 1970 report from the Armed Forces Institute of Pathology described 43 cases of metastatic epididymis lesions. Of these cases, one-third were testicular in origin.[3] Primary sites of disease include the testicle,[3] prostate,[1,2,6-9] stomach,[1-10] pancreas,[12] lymphosarcoma,[2] reticular cell sarcoma,[2] lung,[3] ileum,[13] and colon.[3-5] The mean age of patients presenting with this rare event is 59 years old with a range of 47 to 87 years of age.

Signs and symptoms vary from incidental findings at the time of orchiectomy for metastatic prostate cancer to pain and scrotal swelling in individuals with colon cancer. Metastatic spread to the epididymis may occur in one of four routes according to Willis: 1) Retrograde venous extension; 2) retrograde lymphatic extension; 3) arterial embolism; and 4) direct invasion by neighboring growths.[14]

In previous reports of patients with colon cancer metastatic to the epididymis, widespread disease was also found. Our patient, however, had had resection of a Duke's B sigmoid colon adenocarcinoma three years previously, and after extensive examination, we could find malignant recurrence only at the epididymis. It is interesting to note that in the surgical specimen from our patient, tumor was found in vascular channels in multiple sections. These were endothelial lined tubules which could represent either venous or lymphatic vessels. One could hypothesize a possible route of metastatic seeding via these vessels in this case.

Although metastatic disease to the epididymis is a rare finding, it should nonetheless be considered in the case of an elderly patient with a mass in the epididymis, especially when that patient is known to have had another primary tumor with a cell type capable of seeding metastatic deposits in the epididymis.

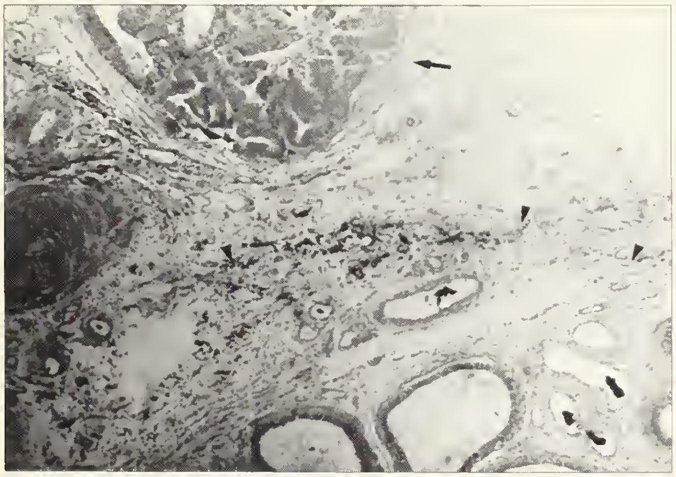

Figure 1
Low Power Photmicrograph. Normal epicidymal tubular structure seen below (arrowheads) and metastatic adenocarcinoma of the colon adjacent (arrow).

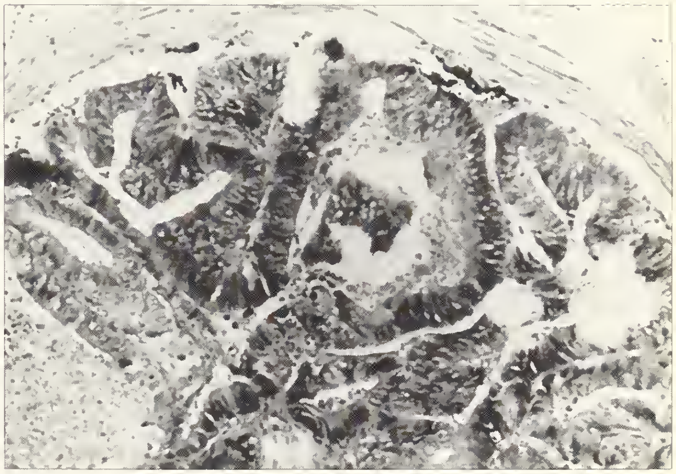

Figure 2
High Power Photomicrograph. Metastatic adenocarcinoma of the colon.

Malignant Tumor of the Colon — Parra et al

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■ **Roger H. Allbee, M.D.,** Festus, Missouri, died February 16, 1992 at age 85. Born in Springfield, IL, Dr. Allbee received his medical degree at the University of Vermont in 1931. He was one of three physicians on staff at Jefferson Memorial Hospital in Crystal City when it opened in 1957. Dr. Allbee was in the medical corps for 26 years, working his way up to colonel. He was awarded the Legion of Merit for service as commanding officer and surgeon at a hospital in Kyoto, Japan during the Korean War. Dr Allbee was a general surgeon in the Crystal City/Festus area for 35 years. He was a member of the International College of Surgeons and the American College of Surgeons.

■ **Charles H. Eller, M.D.,** Santa Fe, NM, died January 26, 1992, at age 88. Dr. Eller graduated from Stanford University and received his medical degree at the University of Colorado, and D.P.H. from Johns Hopkins University in 1930.

■ **Bert H. Klein, M.D.,** Webster Groves, Missouri, died February 24, 1992 at age 81. Dr. Klein received his medical degree at the University of Illinois at Chicago Health Sciences Center, Chicago in 1936. He practiced as a general practioner in St. Louis from 1937 to 1976 and was on the staff of Deaconess Hospital.

■ **William S. Redmond, M.D.,** St. Joseph, Missouri, died January 5, 1992 at age 80. Dr. Redmond received his medical degree at St. Louis University and Rush School of Medicine, Chicago, IL, in 1937. He was a family practioner for 50 years in St. Joseph. Dr. Redmond was a long-time member of Missouri State Medical Association, the Amercian Medical Association, and the American Academy of Family Physicians.

■ **George A. Seib, M.D.,** St. Louis, Missouri, died April 3, 1992, at age 89. Dr. Seib received his medical degree at Washington University, St. Louis, in 1928. He had served 50 years on staff at Lutheran Medical Center and was a former instructor of Anatomy at Washington University.

■ **William F. Utterman, M.D.,** Ste. Genevieve, Missouri, died February 24, 1992 at age 76. Born in LaCrosse, WI, Dr. Utterman attended the University of Utah and obtained his medical degree at Temple University Medical School, Philadelphia, PA, in 1943. During WWII he served in the U.S. Army Medical Corps in Europe as General Hospital and Battalion Surgeon. He joined Perry County Memorial Hospital, Perryville, serving from 1951-1970. Then he became affiliated with Jersey County Community Hospital, Jerseyville, IL. Dr. Utterman served on the staff at Ste. Genevieve Hospital from 1970-1984. He was a member of Perry-Ste. Genevieve County Medical Society, Missouri State Medical Association and American Medical Association.

■ **O. Earl Whitsell, M.D.,** St. Joseph, Missouri, died January 21, 1992 at age 92. Dr. Whitsell received his medical degree at Washington University Medical School in 1924. He was a secretary of Buchanan County Medical Society, and had served as president of staff of the former Missouri Methodist Hospital. He was a member of the American Medical Association, Missouri State Medical Association, Board of Otolaryngology, Board of Ophthalmology, American Academy of Ophthalmology and Otolaryngology, American Association of Ophthalmology, and Fifty Year Club of American Medicine.

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List surgical procedures routinely performed

State(s) where currently licensed

States where formerly licensed (for past 10 years)

Amount of Professional Liability Insurance now carried
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Is the doctor involved with delivery of babies? ☐ YES ☐ NO

02

Kathy Seay, admissions adviser for the University of Missouri -Columbia School of Medicine, hands an envelope containing the residency match for fourth-year medical student Sue Birmingham.

Matching doctors with their futures

March 18th, the 104 students who are graduating in May from the University of Missouri-Columbia School of Medicine, along with other fourth-year medical students across the country, found out how they will spend the next three to eight years.

Gathered in the gallery outside the J. Otto Lottes Health Sciences Library, they opened their envelopes containing their "matches" -- the sites of their postgraduate training program, a requirement for licensure as a physician.

"Match Day probably produces more anxiety and joy than any other single day in a medical student's career," said Robert McCallum, Ph. D., the MU School's assistant dean for student affairs. "At 10:59 you've got a hundred people as anxious as can be. A minute later, you've got screams of relief, shouts of joy and perhaps a few tears shed by the people who are disappointed."

The information contained in the envelopes comes from the National Resident Matching Program, based in Evanston, IL. Last semester, graduating medical students across the country visited residency programs, trying to decide, where they would like to go after medical school. The cost of these trips to the students averaged $1,500.

The students then listed their residency program choices in a computer. The institutions that the students visited also ranked the prospective residents. The rankings were then sent to Evanston. The match the medical students found represents their highest choice that also picked them.

Approximately 16,000 medical students across the country participated in Match Day. Medical schools received word of their students' matches two days early. The information, kept secret until match day, is used to help find matches for students who did not match through the computer. It is hoped by match day that every graduating medical student will have a position and can receive an envelope with the name of their program.

In 1991, 99 percent of MU's seniors participating in the regular match were matched by computer, the highest number in recent memory. This year 96 percent of the MU students matched by computer, compared to the 93 percent average nationally.

Of the 86 students participating at the University of Missouri-Kansas City, 94.1 percent were matched with a resident position. Twenty-eight students will be in residency at Kansas City.

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■ **Susan K. Ailor, M.D.,** 500 Keene, Suite 401, Columbia, MO 65205, has become a member of the Boone County Medical Society. Dr. Ailor received her medical degree at the University of Missouri, Columbia, in 1985. She completed her residency at the University of Missouri, Columbia. Dr. Ailor specializes in dermatology.

■ **Daphne K. Anderson, M.D.,** 1900 S. National, Suite 3800, Springfield, MO 65804, has become a member of the Greene County Medical Society. Dr. Anderson received her medical degree at Tulane University, New Orleans, LA, in 1983. She completed her residency at the University of Missouri, Columbia. Dr. Anderson specializes in dermatology.

■ **Scott J. Arbaugh, M.D.,** 1245 Graham Rd., Florrisant, MO 63031, has become a member of the St. Louis Metropolitan Medical Society. Dr. Arbaugh received his medical degree at St. Louis University in 1985. He completed his residency at Barnes Hospital, Washington University, Department of Psychiatry. Dr. Arbaugh specializes in psychiatry.

■ **Bradley A. Bowenschulte, M.D.,** 1900 S. National Suite 1800, Springfield, MO 65804, has become a member of the Greene County Medical Society. Dr. Bowenschulte received his medical degree at the University of Texas Medical School, Houston, TX, in 1986. He completed his residency at Barnes Hospital, Washington University, St. Louis, MO. Dr. Bowenshulte specializes in anesthesia/pediatrics.

■ **James T. Brocksmith, D.O.,** 500 Keene, Suite 307, Columbia, MO 65205, has become a member of the Boone County Medical Society. Dr. Brocksmith received his medical degree at the University of Osteopathic Medicine, Des Moines, IA, in 1987. He completed his residency at the VA Medical Center, University of Iowa. Dr. Brocksmith specializes in internal medicine.

■ **John W. Cassels, Jr., M.D.,** 1 Hospital Drive, HSCN-623, Columbia, MO 65212, has become a member of the Boone County Medical Society. Dr. Cassels received his medical degree at the University of Florida College of Medicine, Gainesville, FL, in 1979. He completed his residency at Walter Reed Army Medical Center, Washington D.C. Dr. Cassels specializes in obstetrics/gynecology.

■ **George A. Evans, M.D.,** 2703 Clark Lane, Columbia, MO 65205, has become a member of the Boone County Medical Society. Dr. Evans received his medical degree at the University of Missouri, Columbia, in 1983. He completed his residency at The New York Hospital-Cornell, New York, NY. Dr. Evans specializes in anatomic and clinical pathology.

■ **Kathleen M. Garcia, M.D.,** 8000 Bonhomme, Suite 104, Clayton, MO 63105, has become a member of the St. Louis Metropolitan Medical Society. Dr. Garcia received her medical degree at Harvard, Boston, MA, in 1980. She completed her residency at Jewish Hospital, St. Louis. Dr. Garcia specializes in internal medicine.

■ **Robb R. Hicks, IV, M.D.,** 1701 Lacey Emerg. Room, Springfield, MO 63701, has become a member of the Cape Girardeau County Area Medical Society. Dr. Hicks received his medical degree at the University of Southern California, Los Angeles, CA, in 1987. He completed his residency at St. Louis University Hospitals. Dr. Hicks specializes in emergency medicine.

■ **Jeffrey D. Jaax, M.D.,** 1001 E. 101st Terrace, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Jaax received his medical degree at the University of Kansas Medical School in 1987. He completed his residency at the Mayo Clinic, Rochester, MN. Dr. Jaax specializes in anesthesia.

■ **Michael A. Kolda, M.D.,** 1701 Lacey Emerg. Rm., Cape Girardeau, MO 63701, has become a member of the Cape Girardeau County Area Medical Society. Dr. Kolda received his medical degree at the University of Texas Medical Branch, Galveston, TX, in 1981. He completed his residency at Oklahoma University Tulsa Medical College. Dr. Kolda specializes in emergency medicine.

■ **Michael R. Scott, M.D.,** 1900 S. National, Rm. 3400, Springfield, MO 65804, has become a member of the Greene County Medical Society. Dr. Scott received his medical degree at the University of Oklahoma Medical School in 1984. He completed his residency at Oklahoma Memorial Hospital. Dr. Scott specializes in internal medicine.

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NEW MEMBERS

Continued from page 305

- **Kuldeep Singh, M.D.,** 1109 N. Rutheford, Macon 63552, has become a member of the Chariton-Macon-Monroe-Randolph County Medical Society. Dr. Singh received his medical degree at Government Medical College Kashmir University, Srinagar, India, in 1970. He completed his residency at the Booth Memorial Medical Center in Flushing, NY. Dr. Singh specializes in internal medicine.

- **Marc A. Turner, M.D.,** 1001 E. 101st Terrace, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Turner received his medical degree at the University of Kansas Medical Center in 1979. He completed his residency at St. Lukes Hospital, Kansas City, MO. Dr. Turner specializes in anesthesia.

- **Scott A. Turner, M.D.,** Tri-County Clinic Box 407, Osceola, MO 64776, has become a member of the Missouri State Medical Association. Dr. Turner received his medical degree at St. Louis University in 1988. He completed his residency at Cox Medical Centers, Springfield, MO. Dr. Turner specializes in family practice.

- **James M. Walden, Jr., M.D.,** Suite 201, 6650 Troost, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Walden received his medical degree at Baylor College of Medicine, Houston, TX. He completed his residency at the University of Missouri-Kansas City Affiliated Hospitals. Dr. Walden specializes in internal medicine/gastroenterology.

RESIDENT MEMBERS

Sanjay K. Anand, M.D., *Internal Medicine*
Marion H. Baker, M.D., *Internal Medicine*
James A. Birgenheier, Jr., M.D., *Internal Medicine*
John A. Billingsley, III, M.D., *Internal Medicine*
Richard J. Brooks, M.D. , *Internal Medicine*
Shannon L. Casidy, M.D., *Obstetrics/Gynecology*
Tamara E. Crowell, M.D., *Internal Medicine*
Kimberly M. Davies, M.D., *Internal Medicine/Pediatrics*
Judith K. DeGraff, M.D., *General Surgery*
Denise C. Dittemore, M.D., *Internal Medicine*
Darry S. Johnson, M.D., *Internal Medicine*
Nabil K. Kiridly, M.D., *Plastic & Reconstructive Surgery*
Jacqueline Y. Lai, M.D., *Internal Medicine*
Ben D. Lightfoot, M.D., *Internal Medicine*
Nyron T. Marshall, M.D., *Internal Medicine*
Scott A. Nitzel, M.D., *Internal Medicine/Pediatrics*
Eileen M. O'Brien-Su, M.D., *Internal Medicine/Pediatrics*
Timothy J. Ryan, D.O., *Internal Medicine*
Eric M. Ruocco, M.D., *Internal Medicine*
Marcus I. Schmiedt, M.D., *Internal Medicine*
Stephen N. Sides, M.D., *Radiology*
Gregory M. Vardakis, D.O., *Internal Medicine*
Kimberly A. Witkop, M.D., *Internal Medicine*
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Contraindication: Known hypersensitivity to the drug. Because cross sensitivity in this class of compounds has been observed, H_2-receptor antagonists, including Axid, should not be administered to patients with a history of hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

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Drug Interactions—No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently

Carcinogenesis, Mutagenesis, Impairment of Fertility—A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups.The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled ONA synthesis, sister chromatid exchange, mouse lymphoma assay chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of anti-androgenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely

Hematologic—Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

PV 2093 AMP [101591]

Additional information available to the profession on request

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YOCON®

YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it, however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

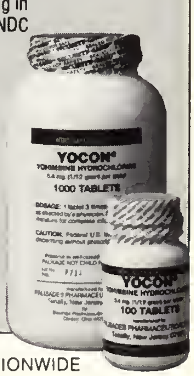
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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Washington University

William A. Peck, M.D., vice chancellor for medical affairs and dean of Washington University School of Medicine in St. Louis, and president of the Washington University Medical Center, has been elected to the rank of fellow by the American Association for the Advancement of Science (AAAS).

Dr. Peck received the honor as it is cited with his nomination, "for distinguished research in bone and mineral metabolism and for leadership in developing an internationally acclaimed center for clinical investigation at Washington University."

Dr. Peck is an expert in the study and treatment of osteoporosis.

Researchers at Washington University will begin study soon on the experimental drug, Lazabemide. The drug will be studied for its safety and effect treating symptoms of Parkinson's Disease.

Washington University is one of 18 medical centers and hospitals across the United States and Canada participating in the research. **Joel S. Perlmutter, M.D.,** associate professor of neurology at Washington University, expects to enroll about 17 subjects.

Volunteers are needed to participate in clinical trails of the drug. Subjects must be newly diagnosed with mild symptoms. It is a double-blind study in which subjects will be randomly assigned to take either a placebo or Lazabemide. They have an 80 percent chance of receiving the medication. Treatment will last up to 13 months.

There is no cost to participate in the study. Subjects in the St. Louis area interested in participating may call Lori McGee-Minnich at 362-7148.

The first study to examine the combined effects of growth hormone and resistance exercise shows that growth hormones do not help muscles grow any faster or operate any more efficiently than exercising alone.

Research instructor, **Kevin Yarasheski, Ph.D.,** looked at 16 men between the ages of 21 and 34. During 12 weeks of heavy resistance training on weight-lifting machines, all major muscle groups were worked on a daily basis. Final measurements discovered no significant difference between the men who had lifted weights and taken the growth hormone and the men who had only lifted.

The only discernible difference between the two groups was in the measurement of fat-free mass. The men who took the growth hormone had less body fat but did not get bigger muscles. Some of their increase in fat-free mass was caused by fluid retention.

Fluid retention may be related to growth hormone side effects. Carpal tunnel compression is caused by pressure on the nerve in the wrist. A couple of cases in this study were reported. Other studies have also found a connection between fluid retention to carpal tunnel syndrome.

Other side effects related to fluid retention are high blood pressure and glucose intolerance.

David G. Russell, Ph.D., associate professor of molecular microbiology at Washington University, has found a modified tuberculosis vaccine useful in the prevention or slowing of the disease leishmaniasis. This disfiguring and potentially deadly parasitic disease afflicts 12 million people worldwide. There have been 25 documented cases of Leishmania infection among U.S. troops who returned with it from the Persian Gulf war.

There are three types of leishmaniasis: cutaneous, visceral and mucocutaneous. The majority Leishmania infections among U.S. soldiers caused cutaneous leishmaniasis, a slow-healing disorder. Some also developed visceral leishmaniasis, which can spread to the bone marrow via infected blood, which is why blood donations are banned from military personnel returning from the Middle East.

The current treatment, pentamidine, requires medical supervision which increases the cost. The cost alone can keep it out of the hands of doctors in the Third World.

Dr. Russel's is an appealing and ultimately inexpensive option. Early results show that the progression of leishmaniasis in mice is slowed by the anti-tuberculosis vaccine BCG (bacillus Calmette-Guerin), modified to contain a gene from the Leishmania parasite.

UNIVERSITY OF MISSOURI-COLUMBIA

Armando R. Favazza, M.D., professor of psychiatry, served as guest editor of *Psychiatric Annals* for a special issue on impulse disorders. The journal included an article by Favazza on repetitive self-mutilation and one about pyromania and firesetting behaviors by **Stephen M. Soltys, M.D.,** MU clinical assistant professor of psychiatry.

Jerry C. Parker, Ph.D., clinical associate professor of psychiatry and a researcher with the Missouri Arthritis Rehabilitation Research Center, recently was named to the Executive Committee of the National Association of Rehabilitation Research and Training Centers. Parker also has been appointed to the Research Committee of the Arthritis Foundation.

Peter Tipton, Ph.D., assistant professor of biochemistry, received a five-year grant from the National Institute of General Medical Sciences for his work on catalytic mechanisms of tartrate dehydrogenase. The enzyme, produced by the bacterium *Pseudomonas putida*, can carry out three different chemical reactions, unlike other bacteria, which can carry out only one reaction.

Karl T. Weber, M.D., professor and chairman of internal medicine, was elected to the Association of University Cardiologists at the group's recent meeting in New Orleans. The association, a scholarly society limits its membership to 125 members considered leaders in academic cardiology.

UNIVERSITY OF MISSOURI-KANSAS CITY

Peter J. Kragel, M.D., recently was named chairman of the Department of Pathology at Truman Medical Center and the University of Missouri-Kansas City School of Medicine, effective July.

Dr. Kragel earned his baccalaureate degree at John Hopkins University, Baltimore, MD, and his medical degree at Georgetown University School of Medicine, Washington, D.C.

Dr. Kragel, currently Vice Chairman of the Pathology Department, joined TMC's staff in 1990 as chief of the Anatomic Pathology Section and director of the Pathology Residency Program. He has also served as director of Laboratories at TMC East and associate professor of Pathology at the UMKC Medical School.

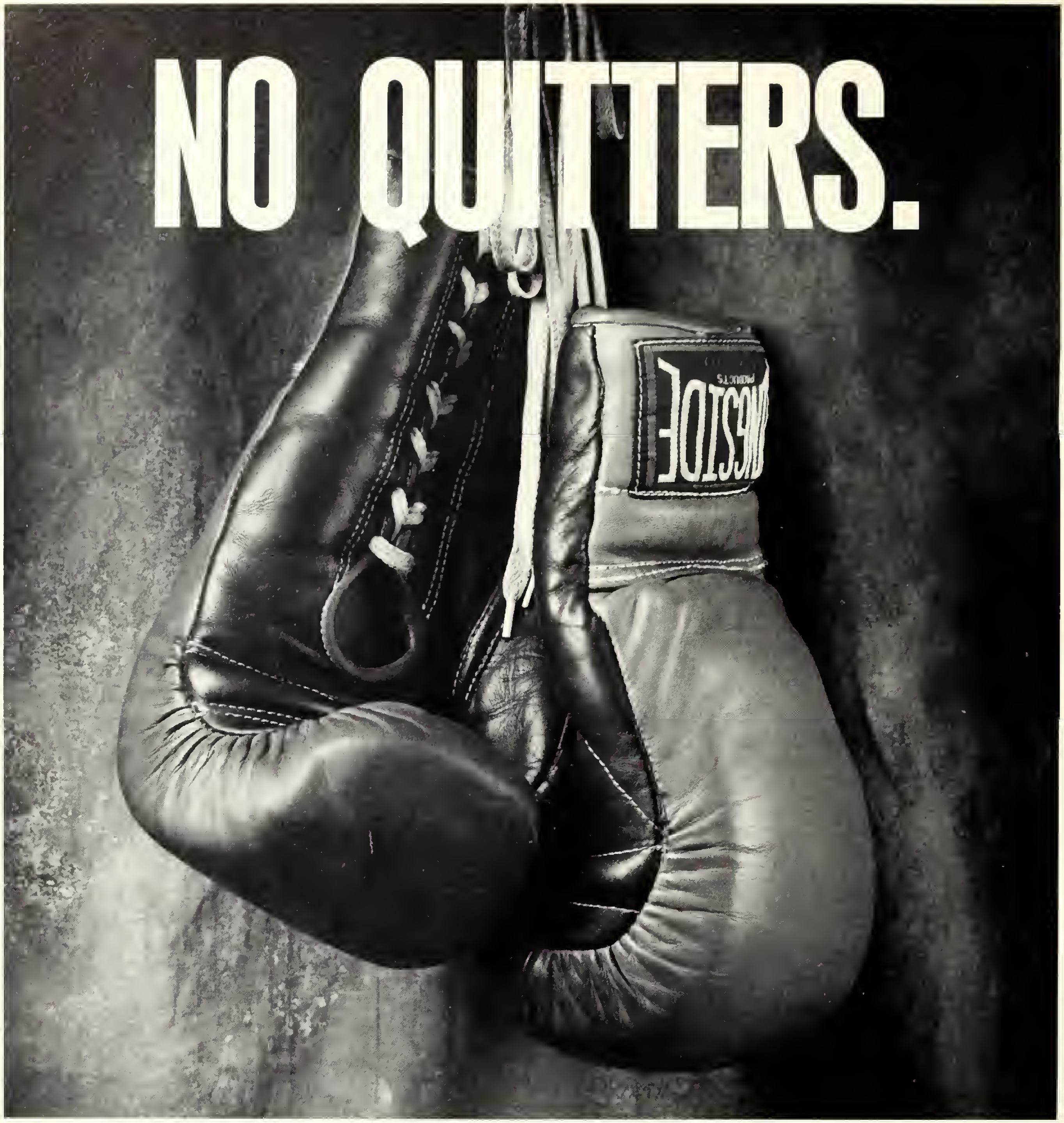
Mitchell Hughey takes a big gulp of air to help resident Regina Selva, M.D., UM-C, listen to his lungs.

A new $400,000 Scanning Transmission Electron Microscope is in place at UMKC. Faculty and graduate student researchers will use it for earlier tumor recognition, more effective microencapsulation in pharmaceutical products and improved dental restorations. A grant from the John W. and Effie E. Speas Memorial Trust and grant monies from the School of Dentistry and Midwest Research Institute made the purchase possible.

UMKC's research focus on aging issues received a major boost through the appointment of **Alfred F. Esser, Ph.D.,** as the Marion Merrell Dow professor in the School of Basic Life Sciences. He is conducting research that could one day determine causes of premature aging.

Dr. Esser was professor and associate chairman of research at the Department of Comparative and Experimental Pathology at the University of Florida, Gainesville. His appointment is the first of two endowed chairs that are a part of the Scientific Education Partnership, a collaboration enterprise between the Marion Merrell Dow Foundation, UMKC and the University of Kansas to study issues of aging.

Dr.Esser's research will focus on the "program theory of aging," which states that an intentional sequence of events written in the genetic makeup leads to age changes. These same events are required for the necessary elimination of unwanted cells in the development of the immune and nervous system.



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May 1992

1 - *St. Louis*: 2nd William Harkins Lectureship. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

2 - *St. Louis*: 3rd Annual Symposium on Hypercholesterolemia: Practical Aspects of Patient Management. 3 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

5 & 6 - *St. Louis*: Transesophageal Echocardiography Course. 13 credit hours. Contact Dianne Reid, St. Louis University Medical Center, 314/577-8897.

7 to 8 - *Kansas City*: Surgical Symposium - Advances in Surgical Care. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

7 to 9 - *Topeka, KS*: Intermediate & Advanced Workshops in Hypnotherapy. 14 credit hours. Contact Division of Continuing Education, The Menninger Clinic, Box 829, Topeka, KS 66601-0829. Fax: 913/273/9941, 800/288/7377.

7 to 10 - *St. Petersburg, FL*: Emergency Department Legal Environment: Medicine's Response. 11 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

8 - *Kansas City*: Office Evaluation of Sleep Disorders. Category 1 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

8 - *Kansas City:* Practical Cardiology. 6 credit hours. Contact the Ed Bixby Institue for Postgraduate Medical Education, Research Medical Center, 2316 E. Meyer Blvd., Kansas City, MO 64132. PH 816/276-4306.

11 - *St. Louis*: Interventional Radiology. A city-wide radiology conference by Daniel Picus, M.D. Contact R. Gilbert Jost, M.D, Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

12 - *St. Louis*: Pediatric Advanced Cardiac and Trauma Support & Pediatric Advanced Life Support Recert. Course. 7 credit hours. Contact Mary Beth Foerstel, R.N., St. Louis University Medical Center, 314/577-5385.

13 - *St. Louis*: Update on Summer Sports and Sports Medicine. Contact Judith L. O'Guin, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141, 314/569-6290.

13 to 15 - *Kansas City*: Coronary Angioplasty for the Basic and Intermediate Dilator. Category 1 credit. Contact Beth M. Paul, Conference Coordinator, CME, St. Luke's Hospital. 816/932-2301.

14 & 15 - *Kansas City*: 24th Annual Matt Weis Symposium - Advances in Obstetrics & Gynecology. Contact Judith L. O'Guin, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141, 314/569-6290.

15 & 16 - *St. Louis*: Advanced Laparoscopy for the General Surgeon. Catagory 1 credit and endorsed by the SAGES. Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 South Euclid, St. Louis, MO 63110-1093. PH: 314/362-6893 or 800/325-9862.

15 to 17 - *St. Joesph*: Advanced Cardiac Life Support Provider Course. 22 credit hours. Contatct Barb Tunks, R.N., Heartland Health System Critical Care Educator, 801 Faraon, St. Joesph, MO 64501, 816/271-7859.

15 to 19 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop: Advanced Microsurgery of the Lumbar & Cervical Spine. 36 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

18 to 22 - *St. Louis*: Body CT Program. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

18 to 23 - *St. Louis*: Health Care Ethics in a Pluralistic Society. 43 credit hours. Contact Donna Troy, St. Louis University Medical Center, 314/577-8195.

27 to 29 - *Kansas City*: Annual Update in OB/GYN. Jointly sponsored with Truman Medical Center. 16 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

30 - *St. Louis*: Systemic Embolization & Stroke. 3.25 credit hours. Contact Office of CME, Washington University School of Medicine, Box 8063, 660 S. Euclid, St. Louis, MO 63110-1093. 314/362-6893 or 800/325-9862.

June 1992

2 to 4 - *St. Louis*: Doppler Echocardiography- The Quantitative Approach. 20 credit hours. Contact Dianne Reid, St. Louis University Medical Center, 314/577-8897.

3 - *St. Louis*: Pulmonary Embolus in Thromembolic Disease. Contact Judith L. O'Guin, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141, 314/569-6290.

Continued from page 313

5 - *Kansas City*: Prostrate Cancer. Contact The Ed Bixby Institute for Postgraduate Medical Education, 2316 E. Meyer Blvd., Kansas City, MO 64132, 816/276-4306 or 800/892-5771.

4 to 6 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part I: Peripheral Nerve. 22 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

4 to 7 - *St. Louis*: Advances in Aesthetic & Reconstructive Breast Surgery. Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 S. Euclid, St. Louis, MO 63110-1093. PH 800/325-9862, 314/362-6893.

6 - *Kansas City*: Fourth Annual Lipid Conference. Jointly sponsored with UMKC School of Medicine. 3 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

6 - *St. Louis*: New Concepts in the Management of Parkinson's Disease. 5 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

7 to 9 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part II: Spine-Microsurgery & Stabilization. 24 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

8 to 12 - *St. Louis*: Neuro CT Program. 25 credit hours. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

10 - *Kansas City*: Domestic Violence Awareness. 3 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

10 to 14 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part III: Microsurgery of the Brain. 36 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

11 to 13 - *St. Louis*: Cornea & Contact Lens Course. Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 South Euclid, St. Louis, MO 63110-1093. PH: 314/362-6893 or 800/325-9862.

12 to 13 - *Columbia, MO*: Common Childhood Problems - For the Practitioner Caring for Children. Contact Barry King, University of Missouri-Columbia School of Medicine, Office of CME, MA215 Health Sciences Center, Columbia, MO 65212. PH 314/822-4105.

15 to 17 - *Athens, Greece*: International Conference on Ischemic Syndromes. 16 credit hours.Contact Ralf Lucus, St. Louis University Medical Center, 314/577-8811.

15 to 19 - *St. Louis*: Body CT Program. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

16 to 18 - *St. Louis*: St. Louis Summer Geriatric Institute. 23 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

22 to 26 - *St. Louis*: Magnetic Resonance Imaging of Head & Body. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

25 to 27 - *St. Louis*: Pediatric Advanced Cardiac & Trauma Support & Pediatric Advanced Life Support Provider Course. 20 credit hours. Contact Mary Beth Foerstel, R.N., St. Louis University Medical Center, 314/577-5385.

26 to 28 - *St. Louis*: Frontiers in Endourology: Laparoscopic Nephrectomy and Beyond. (Urologists Only) Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 South Euclid, St. Louis, MO 63110-1093. PH: 314/362-6893 or 800/325-9862.

July 1992

10 & 11 - *St. Louis*: Advanced Trauma Life Support Course. 16 credit hours.Contact Mary Keegan, R.N., St. Louis University Medical Center, 314/577-5385.

11 to 18 - *London, England & Lyon, France*: Southern Association for Geriatric Medicine 2nd Annual Meeting. For more information call the Southern Association for Geriatric Medicine, 800/423-4992 or 205/945-8425.

15 to 17 - *St. Louis*: Pediatric Advanced Cardiac & Trauma Support & Pediatric Advanced Life Support Provider Course. 20 credit hours. Contact Mary Beth Foerstel, R.N., St. Louis University Medical Center, 314/577-5385.

18 - *St. Louis*: Smoking Cessation. 4 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

28 & 29 - *St. Louis*: Transesophageal Echocardiography Course. Contact Dianne Reid, St. Louis University Medical Center, 314/577-8897.

31 to Aug. 1 - *St. Louis*: Advanced Cardiac Life Support Provider/Recertification. Course. 16 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

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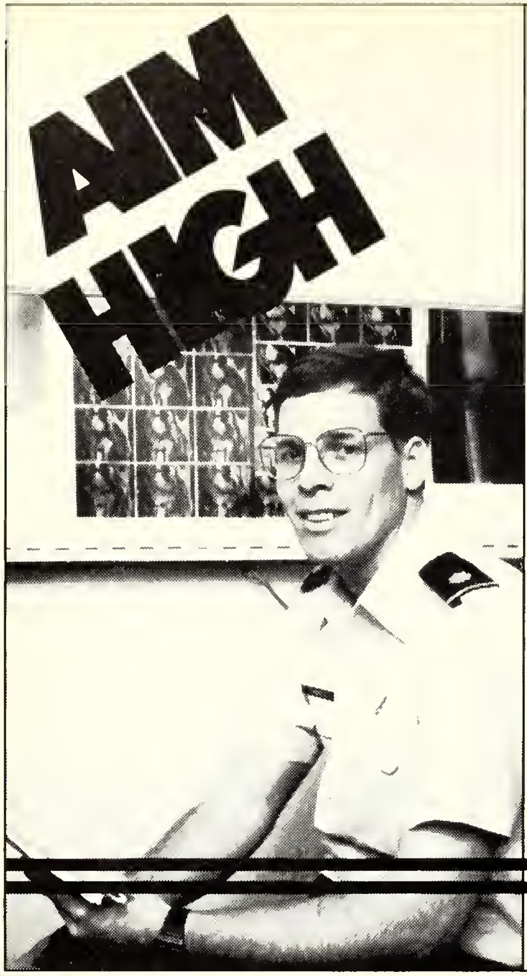

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‡Verapamil should be administered cautiously to patients with impaired renal function.

References: 1. Data on file, Searle. 2. Edmonds D, Würth JP, Baumgart P, et al. Twenty-four-hour monitoring of blood pressure during calcium antagonist therapy. In: Fleckenstein A, Laragh SH, eds. *Hypertension—the Next Decade: Verapamil in Focus*. New York, NY: Churchill Livingstone; 1987:94-100. 3. Midtbø KA. Effects of long-term verapamil therapy on serum lipids and other metabolic parameters. *Am J Cardiol*. 1990;66:13I-15I. 4. Fagher B, Henningsen N, Hulthén L, et al. Antihypertensive and renal effects of enalapril and slow-release verapamil in essential hypertension. *Eur J Clin Pharmacol*. 1990;39(suppl 1):S41-S43. 5. Schmieder RE, Messerli FH, Garavaglia GE, et al. Cardiovascular effects of verapamil in patients with essential hypertension. *Circulation*. 1987;75:1030-1036. 6. Midtbø K, Lauve O, Hals O. No metabolic side effects of long-term treatment with verapamil in hypertension. *Angiology*. 1988;39:1025-1029.

BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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Journal of the Missouri State Medical Association June 1992

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- *Kayaking as a Risk Factor for Leptospirosis*
- *Aggressive Rehabilitation Following Anterior Cruciate Reconstruction*
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The subscription price per year for nonmembers is $20.00. The subscription price for members is included in the annual membership dues. The international subscription price is $40.00. Single copies are $4.00 and the cost of the annual August Directory issue is $15.00.

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Printed by the Ovid Bell Press, Inc., Fulton, Missouri 65251.

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MONETAMED PROFILE

THOMAS P. DOYLE, M.D.

HOME: Springfield, Missouri.

AGE: 36.

SPECIALTY: Orthopedic Surgery.

MEDICAL COLLEGE: University of Missouri, 1978

PRACTICE: "I recently purchased an established but declining sole practitioner office from a retiring physician. It's a risk, but they say that great deeds are usually wrought at great risk."

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Journal of the Missouri State Medical Association

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THE JUNE COVER: Robert D. Shaw, M.D., Willow Springs, MO, the author of "Kayaking as a Risk Factor for Leptospirosis", provides this month's cover art as well. Dr. Shaw made this photo using a 200 mm zoom lens on a Nikon FE. The kayaker is Jed Schlagel and he is running Sock-Em-Dog rapid on the Chattooga River in Georgia.

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Taking the guesswork out of bloodborne pathogen regulations?

J. Regan Thomas, M.D., Editor

The new Bloodborne Pathogen Regulations imposed by OSHA are widely inclusive, unequivocal, **and** they are here now.

The standards went into effect March 6, 1992 and are set to be phased in over a five month period. The initial phase, the so called " employer's exposure control plan," should have been in place by May 5. The next phase which mandates that your office has established employee training programs and an individual office record-keeping system for the regulations, must be in place by June 4, 1992. By July 6, 1992 specific activities within the office environment must have controls and policies in place including personal protective equipment and clothing, policies for lab and patient areas and housekeeping. Employees must have HBV vaccine available. Certain labeling and signage in the office must be in place. I know you can guess who pays for all of these activities.

You may not have guessed however, how extensive these regulations can be. Regardless of your level of enthusiasm for these new guidelines implementation is critical. OSHA has the ability apparently to impose penalties up to $70,000 for each willful or repeated violation, with $7,000 a day for each day the violation continues beyond an established date. Those are penalty ceilings, but there is a minimum penalty for willful violations of $5,000. It gets your attention doesn't it.

Most physician offices will be affected by these regulations. If there is any risk of exposure to blood or potentially infectious materials to any personnel, these regulations will come into effect. This includes not only surgical or procedure oriented activities, but laundry, drapes, dressings and cultures. Exposure not only means needle sticks or cuts, but also any potential contact of infectious material or blood with the eyes, mouth or mucous membranes.

You guessed correctly if you assumed that you as the employer are required to also provide all personal protective equipment and clothing for employees. Likewise you are responsible for cleaning all this equipment and laundering clothing without expense to the employee (including repair or replacement as needed). You must ensure that your employees are using this protective equipment and document an investigation if they are not.

Regulations also stipulate that training programs be provided during normal working hours for all employees. Records must be kept documenting employee training, incidents of exposure, updated exposure control plans, as well as other administrative activities to comply with the regulations. You must provide HBV vaccine at no charge to employees. If exposure occurs, the employee must be provided with documented counseling and medical follow-up.

Now all of this sounds complicated, so you probably guessed that OSHA has provided material to assist you with compliance. As I gathered material to write this editorial I called the OSHA number listed for "more information and assistance." Bureaucracy as only Washington D.C. can foster was in full bloom. I was routed through three individuals at that office before I finally was referred to Dr. Susan Harwood. Dr. Harwood unfortunately was not available. Her secretary said I should have called the OSHA regional office anyway and gave me the number in Kansas City to call. I called the regional office (I'm now meeting OSHA person number five on my telephone trek) and informed that I should call my local office in St. Louis. At least this would be a local call. Though I wasted considerable time calling the suggested number on the OSHA materials, it was nice to know that in hard economic times OSHA was doing its part to provide employment. I called the sixth OSHA person as sug-

Continued on page 328

YOCON®
YOHIMBINE HCl

Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

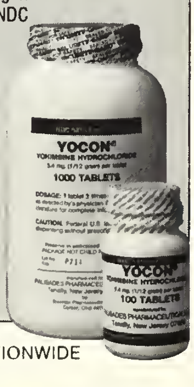
Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

Rev. 1/85



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Editor's Desk

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gested.

The St. Louis office was courteous and the individual on rotation to take the call was willing to talk. I told him about my telephone adventures starting with the initial call for "more information and assistance." He seemed surprised I didn't know to call the Department of Labor, listed in the St. Louis directory white pages under Government Service and ask to be referred to the office for Occupational Safety and Health administration. As you guessed, I'm still new to this bureaucracy protocol.

The inside information I got was that more scrutiny likely would be placed on larger employers than small individual practice settings when random inspections begin. The exception would be if a specific employee complaint was registered. OSHA has usually considered the type of violation, the history and present attempts at compliance, when assessing penalties. If you have not started your exposure control plan yet, you are not in compliance and at risk. If you have individual questions about your plan, you may not want to spend time calling the "suggested" OSHA telephone number. Call your local office instead.

What is the next government activity to take place in your practice? You can only guess.

■

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LETTERS TO THE EDITOR

Dear Editor:

"Fast, Good, and Cheap" As the saying goes, there is fast, there is good, and there is cheap. Pick any two.

Regardless of "rights" or the laws, all three are not likely to happen simultaneously. In fact, with health care even "good" is not enough. People are demanding fast, cheap, and the best health care. We can deliver fast, we can deliver cheap, and we can deliver the best, but not all three at once (and additionally be liable for all adverse outcomes).

In this age of total abdication of individual responsibility, the sad truth is that the individual has much more input into what his or her health will be than the "system."

How many liver transplants are you going to give an alcoholic? How many lung transplants to a chain smoker? Pick a major illness or disease that is costing society billions of dollars and odds are that it is linked to individual behavior. You can regulate the doctors until they can't get anything done and it will not change the above.

Many would have us believe that the health care system is primarily physician driven. I seriously doubt it. Cheap care is still available. We are constantly being compared with what care used to cost in the "good old days." Well, we can still deliver that cheap care. One does not have to go on a dialysis machine. While he or she was in the White House, President and Mrs. John F. Kennedy had a premature baby. Its care did not cost much, but it lived only a few days. I could go on with hundreds of examples. Grandmother does not have to be put in a nursing home. In the nursing home, if she has ice cream for dessert, it is an additional health care expense. If overnight the legislators and lawyers would protect us from the consequences, we could drastically cut health care expenses by going back and delivering the old style inexpensive medicine. There would be a quality problem but if people truly want cheap care, they can have it.

However, people do not truly want just cheap care. They want the best, and they want it fast. People used to have a rudimentary understanding that it is difficult to "have it all." No more, not in this age of third party payers, the expanded government role in medicine, the illusion of a free lunch, and unlimited "entitlements." To quote Yogi Berra, "The future ain't what it used to be."

Ed Masters, M.D.
Cape Girardeau

Dear Editor:

Recently a physician wrote to *Missouri Medicine* alleging that the Board of Healing Arts was taking discipline on BNDD complaints, not protecting the public. This assertion concerns us in several particulars.

The facts are that BNDD complaints represented only 11% of the complaints received in 1991. The percentage is 7% so far for 1992.

The larger issue, however, is that the Board believes that such disciplines DO represent important public protection actions. Let me cite several examples:

In one case the Board disciplined a physician for prescribing several thousand dosage units of a scheduled narcotic to a patient with little or nothing in the record to support his diagnosis.

In another case, the Board disciplined a physician for writing a prescription for a Schedule II amphetamine when his partner asked for a "pick- me-up". There was no medical record of evidence of a physical examination.

The Board disciplined another physician who wrote pre-signed prescriptions for his office nurse who was seriously abusing drugs. These scripts were written over a significant number of years.

The Board feels that these cases and a number of others represented a threat to quality of care for the public. The Board felt that they were of such seriousness that the Board's obligation to the public demanded more than a simple admonition to the physician to stop these practices. Thus the Board felt that the cases represented important consumer protection action.

In this magazine a physician recently charged that an elderly physician was disciplined for writing a refill of a prescription for his terminally ill wife, written originally by her treating physician. These assertions are simply not true. The physician was not disciplined. The Board, on a completely confidential basis, did advise the physician that a better practice would have been to get another physician to see her if her treating physician could not be reached. We use such a confidential "letter of concern" where there is no evidence of a violation but where the Board is concerned about some aspect of the case. This happens in about 20% of our cases.

Frederick A. Tromans, Executive Director,
Board of Registration for the Healing Arts

Revised Cost estimates:

Dr. Charles Norland's article, "Liability Epidemic - Health Care Costs: Break The Linkage, (April 1992), included estimates that the medical profession spends $25 billion a year on malpractice premiums and that the cost of defensive medicine adds $21 billion to the United States care bill each year. Dr. Norland's sources now report revised estimates of $7 and $15 billion for malpractice premiums and defensive medicine each year.

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quinapril HCl tablets 10, 20, 40 mg

* See DOSAGE AND ADMINISTRATION section of prescribing information.
† If, after an adequate trial of ACCUPRIL alone, based on your medical judgment as the prescribing physician, you determine that your patient requires the addition of a diuretic, Parke-Davis will refund to the patient his/her cost for the diuretic prescription less any amount reimbursed or paid for by an HMO, insurance company, or any other plan or program.

For more details, ask your Parke-Davis Representative or call 1-800-955-3077.

‡ In some patients, the antihypertensive effect may diminish toward the end of the once-daily dosing interval. In such patients, an increase in dosage or twice-daily administration may be warranted.

ACCUPRIL is available in 10, 20, and 40 mg tablets. Usual initial starting dosage is 10 mg once daily.

ACCUPRIL is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

Please see brief summary of prescribing information on following page.

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PARKE-DAVIS

> **USE IN PREGNANCY**
>
> When used in pregnancy during the second and third trimesters, ACE inhibitors can cause injury and even death to the developing fetus. When pregnancy is detected, ACCUPRIL should be discontinued as soon as possible. See WARNINGS, Fetal/Neonatal Morbidity and Mortality

Before prescribing, please see full prescribing information. A brief summary follows.

INDICATIONS AND USAGE

ACCUPRIL is indicated for the treatment of hypertension. It may be used alone or in combination with thiazide diuretics.

In using ACCUPRIL, consideration should be given to the fact that another angiotensin-converting enzyme (ACE) inhibitor, captopril, has caused agranulocytosis, particularly in patients with renal impairment or collagen vascular disease. Available data are insufficient to show that ACCUPRIL does not have a similar risk (see WARNINGS).

CONTRAINDICATIONS

ACCUPRIL is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an ACE inhibitor.

WARNINGS

Angioedema: Angioedema of the face, extremities, lips, tongue, glottis, and larynx has been reported in patients treated with ACE inhibitors and has been seen in 0.1% of patients receiving ACCUPRIL. Angioedema associated with laryngeal edema can be fatal. If laryngeal stridor or angioedema of the face, tongue, or glottis occurs, treatment with ACCUPRIL should be discontinued immediately, the patient treated in accordance with accepted medical care, and carefully observed until the swelling disappears. In instances where swelling is confined to the face and lips, the condition generally resolves without treatment, antihistamines may be useful in relieving symptoms. **Where there is involvement of the tongue, glottis, or larynx likely to cause airway obstruction, emergency therapy including, but not limited to, subcutaneous epinephrine solution 1:1000 (0.3 to 0.5 mL) should be promptly administered (see ADVERSE REACTIONS).**

Hypotension: Symptomatic hypotension was rarely seen in uncomplicated hypertensive patients treated with ACCUPRIL but, as with other ACE inhibitors, it is a possible consequence of therapy in salt/volume depleted patients, such as those previously treated with diuretics or dietary salt restriction or who are on dialysis (see PRECAUTIONS, DRUG INTERACTIONS, and ADVERSE REACTIONS). In controlled studies, syncope was observed in 0.4% of patients (N = 3203), this incidence was similar to that observed for captopril (1%) and enalapril (0.8%).

In patients with concomitant congestive heart failure, with or without associated renal insufficiency, ACE inhibitor therapy may cause excessive hypotension, which may be associated with oliguria or azotemia and, rarely, with acute renal failure and death. In such patients, ACCUPRIL therapy should be started at the recommended dose under close medical supervision. These patients should be followed closely for the first 2 weeks of treatment and whenever the dosage of antihypertensive medication is increased (see DOSAGE AND ADMINISTRATION).

If symptomatic hypotension occurs, the patient should be placed in the supine position and, if necessary, normal saline may be administered intravenously. A transient hypotensive response is not a contraindication to further doses; however, lower doses of ACCUPRIL or reduced concomitant diuretic therapy should be considered.

Neutropenia/Agranulocytosis: Another ACE inhibitor, captopril, has been shown to cause agranulocytosis and bone marrow depression rarely in patients with uncomplicated hypertension, but more frequently in patients with renal impairment, especially if they also have a collagen vascular disease such as systemic lupus erythematosus or scleroderma. Agranulocytosis did occur during ACCUPRIL treatment in one patient with a history of neutropenia during previous captopril therapy. Available data from clinical trials of ACCUPRIL are insufficient to show that, in patients without prior reactions to other ACE inhibitors, ACCUPRIL does not cause agranulocytosis at similar rates. As with other ACE inhibitors, periodic monitoring of white blood cell counts in patients with collagen vascular disease and/or renal disease should be considered

Fetal/Neonatal Morbidity and Mortality: ACE inhibitors can cause fetal and neonatal morbidity and death when administered to pregnant women. Several dozen cases have been reported in the world literature. When pregnancy is detected, ACE inhibitors should be discontinued as soon as possible.

The use of ACE inhibitors during the second and third trimesters of pregnancy has been associated with fetal and neonatal injury, including hypotension, neonatal skull hypoplasia, anuria, reversible or irreversible renal failure, and death. Oligohydramnios has also been reported, presumably resulting from decreased fetal renal function, oligohydramnios in this setting has been associated with fetal limb contractures, craniofacial deformation, and hypoplastic lung development. Prematurity, intrauterine growth retardation, and patent ductus arteriosus have also been reported, although it is not clear whether these occurrences were due to the ACE inhibitor exposure.

ONCE-A-DAY*
ACCUPRIL® ™
quinapril HCl tablets

These adverse effects do not appear to have resulted from intrauterine ACE inhibitor exposure that has been limited to the first trimester. Mothers whose embryos and fetuses are exposed to ACE inhibitors only during the first trimester should be so informed. Nonetheless, when patients become pregnant, physicians should make every effort to discontinue the use of ACCUPRIL as soon as possible.

Rarely (probably less often than once in every thousand pregnancies), no alternative to ACE inhibitors will be found. In these rare cases, the mothers should be apprised of the potential hazards to their fetuses, and serial ultrasound examinations should be performed to assess the intraamniotic environment.

If oligohydramnios is observed, ACCUPRIL should be discontinued unless it is considered life-saving for the mother. Contraction stress testing (CST), a non-stress test (NST), or biophysical profiling (BPP) may be appropriate, depending upon the week of pregnancy. Patients and physicians should be aware, however, that oligohydramnios may not appear until after the fetus has sustained irreversible injury.

Infants with histories of *in utero* exposure to ACE inhibitors should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion. Exchange transfusion or dialysis may be required as a means of reversing hypotension and/or substituting for disordered renal function. Removal of ACCUPRIL, which crosses the placenta, from the neonatal circulation is not significantly accelerated by these means.

No teratogenic effects of ACCUPRIL were seen in studies of pregnant rats and rabbits. On a mg/kg basis, the doses used were up to 180 times (in rats) and one time (in rabbits) the maximum recommended human dose.

PRECAUTIONS

General

Impaired renal function: As a consequence of inhibiting the renin-angiotensin-aldosterone system, changes in renal function may be anticipated in susceptible individuals. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with ACE inhibitors, including ACCUPRIL, may be associated with oliguria and/or progressive azotemia and rarely acute renal failure and/or death.

In clinical studies in hypertensive patients with unilateral or bilateral renal artery stenosis, increases in blood urea nitrogen and serum creatinine have been observed in some patients following ACE inhibitor therapy. These increases were almost always reversible upon discontinuation of the ACE inhibitor and/or diuretic therapy. In such patients, renal function should be monitored during the first few weeks of therapy.

Some hypertensive patients with no apparent preexisting renal vascular disease have developed increases in blood urea and serum creatinine, usually minor and transient, especially when ACCUPRIL has been given concomitantly with a diuretic. This is more likely to occur in patients with preexisting renal impairment. Dosage reduction and/or discontinuation of any diuretic and/or ACCUPRIL may be required.

Evaluation of hypertensive patients should always include assessment of renal function (see DOSAGE AND ADMINISTRATION).

Hyperkalemia and potassium-sparing diuretics: In clinical trials, hyperkalemia (serum potassium ≥5.8 mmol/L) occurred in approximately 2% of patients receiving ACCUPRIL. In most cases, elevated serum potassium levels were isolated values which resolved despite continued therapy. Less than 0.1% of patients discontinued therapy due to hyperkalemia. Risk factors for the development of hyperkalemia include renal insufficiency, diabetes mellitus, and the concomitant use of potassium-sparing diuretics, potassium supplements, and/or potassium-containing salt substitutes, which should be used cautiously, if at all, with ACCUPRIL (see PRECAUTIONS, Drug Interactions).

Cough: Cough has been reported with the use of ACE inhibitors. Characteristically, the cough is nonproductive, persistent, and resolves after discontinuation of therapy. ACE inhibitor-induced cough should be considered as part of the differential diagnosis of cough.

Surgery/anesthesia: In patients undergoing major surgery or during anesthesia with agents that produce hypotension, ACCUPRIL will block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Information for Patients

Pregnancy: Female patients of childbearing age should be told about the consequences of second- and third-trimester exposure to ACE inhibitors, and they should also be told that these consequences do not appear to have resulted from intrauterine ACE-inhibitor exposure that has been limited to the first trimester. These patients should be asked to report pregnancies to their physicians as soon as possible.

Angioedema: Angioedema, including laryngeal edema, can occur with treatment with ACE inhibitors, especially following the first dose. Patients should be so advised and told to report immediately any signs or symptoms suggesting angioedema (swelling of face, extremities, eyes, lips, tongue, difficulty in swallowing or breathing) and to stop taking the drug until they have consulted with their physician (see WARNINGS).

Symptomatic hypotension: Patients should be cautioned that lightheadedness can occur, especially during the first few days of ACCUPRIL therapy, and that it should be reported to a physician. If actual syncope occurs, patients should be told to not take the drug until they have consulted with their physician (see WARNINGS).

All patients should be cautioned that inadequate fluid intake or excessive perspiration, diarrhea, or vomiting can lead to an excessive fall in blood pressure because of reduction in fluid volume, with the same consequences of lightheadedness and possible syncope.

Patients planning to undergo any surgery and/or anesthesia should be told to inform their physician that they are taking an ACE inhibitor.

Hyperkalemia: Patients should be told not to use potassium supplements or salt substitutes containing potassium without consulting their physician (see PRECAUTIONS).

Neutropenia: Patients should be told to report promptly any indication of infection (eg, sore throat, fever) which could be a sign of neutropenia.

NOTE: As with many other drugs, certain advice to patients being treated with ACCUPRIL is warranted. This information is intended to aid in the safe and effective use of this medication. It is not a disclosure of all possible adverse or intended effects.

Drug Interactions

Concomitant diuretic therapy: As with other ACE inhibitors, patients on diuretics, especially those on recently instituted diuretic therapy, may occasionally experience an excessive reduction of blood pressure after initiation of therapy with ACCUPRIL. The possibility of hypotensive effects with ACCUPRIL may be minimized by either discontinuing the diuretic or cautiously increasing salt intake prior to initiation of treatment with ACCUPRIL. If it is not possible to discontinue the diuretic, the starting dose of quinapril should be reduced (see DOSAGE AND ADMINISTRATION).

Agents increasing serum potassium: Quinapril can attenuate potassium loss caused by thiazide diuretics and increase serum potassium when used alone. If concomitant therapy of ACCUPRIL with potassium-sparing diuretics (eg, spironolactone, triamterene, or amiloride), potassium supplements, or potassium-containing salt substitutes is indicated, they should be used with caution along with appropriate monitoring of serum potassium (see PRECAUTIONS).

Tetracycline and other drugs that interact with magnesium: Simultaneous administration of tetracycline with ACCUPRIL reduced the absorption of tetracycline by approximately 28% to 37%, possibly due to the high magnesium content in ACCUPRIL tablets. This interaction should be considered if coprescribing ACCUPRIL and tetracycline or other drugs that interact with magnesium.

Lithium: Increased serum lithium levels and symptoms of lithium toxicity have been reported in patients receiving concomitant lithium and ACE inhibitor therapy. These drugs should be co-administered with caution, and frequent monitoring of serum lithium levels is recommended. If a diuretic is also used, it may increase the risk of lithium toxicity.

Other agents: Drug interaction studies of ACCUPRIL with other agents showed:

- Multiple dose therapy with propranolol or cimetidine has no effect on the pharmacokinetics of single doses of ACCUPRIL.
- The anticoagulant effect of a single dose of warfarin (measured by prothrombin time) was not significantly changed by quinapril coadministration twice-daily.
- ACCUPRIL treatment did not affect the pharmacokinetics of digoxin.
- No pharmacokinetic interaction was observed when single doses of ACCUPRIL and hydrochlorothiazide were administered concomitantly.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Quinapril hydrochloride was not carcinogenic in mice or rats when given in doses up to 75 or 100 mg/kg/day (50 to 60 times the maximum human daily dose, respectively, on a mg/kg basis and 3.8 to 10 times the maximum human daily dose when based on a mg/m² basis) for 104 weeks. Female rats given the highest dose level had an increased incidence of mesenteric lymph node hemangiomas and skin/subcutaneous lipomas. Neither quinapril nor quinaprilat were mutagenic in the Ames bacterial assay with or without metabolic activation. Quinapril was also negative in the following genetic toxicology studies: *in vitro* mammalian cell point mutation, sister chromatid exchange in cultured mammalian cells, micronucleus test with mice, *in vitro chromosome* aberration with V79 cultured lung cells, and in an *in vivo* cytogenetic study with rat bone marrow. There were no adverse effects on fertility or reproduction in rats at doses up to 100 mg/kg/day (60 and 10 times the maximum daily human dose when based on mg/kg and mg/m², respectively).

Pregnancy

Pregnancy Categories C (first trimester) and D (second and third trimesters): See WARNINGS, Fetal/Neonatal Morbidity and Mortality

Nursing Mothers

It is not known if quinapril or its metabolites are secreted in human milk. Quinapril is secreted to a limited extent, however, in milk of lactating rats (5% or less of the plasma drug concentration was found in rat milk). Because many drugs are secreted in human milk, caution should be exercised when ACCUPRIL is given to a nursing mother.

Geriatric Use

Elderly patients exhibited increased area under the plasma concentration time curve (AUC) and peak levels for quinaprilat compared to values observed in younger patients; this appeared to relate to decreased renal function rather than to age itself. In controlled and uncontrolled studies of ACCUPRIL where 918 (21%) patients were 65 years and older, no overall differences in effectiveness or safety were observed between older and younger patients. However, greater sensitivity of some older individual patients cannot be ruled out.

Pediatric Use

The safety and effectiveness of ACCUPRIL in children have not been established.

ADVERSE REACTIONS

ACCUPRIL has been evaluated for safety in 4960 subjects and patients. Of these, 3203 patients, including 655 elderly patients, participated in controlled clinical trials. ACCUPRIL has been evaluated for long-term safety in over 1400 patients treated for 1 year or more.

Adverse experiences were usually mild and transient.

Discontinuation of therapy because of adverse events was required in 4.7% of patients treated with ACCUPRIL in placebo-controlled hypertension trials.

Adverse experiences probably or possibly related to therapy or of unknown relationship to therapy occurring in 1% or more of the 1563 patients in placebo-controlled hypertension trials who were treated with ACCUPRIL are shown below.

Adverse Events in Placebo-Controlled Trials

	ACCUPRIL (N = 1563) Incidence (Discontinuance)	Placebo (N = 579) Incidence (Discontinuance)
Headache	5.6 (0.7)	10.9 (0.7)
Dizziness	3.9 (0.8)	2.6 (0.2)
Fatigue	2.6 (0.3)	1.0
Coughing	2.0 (0.5)	0.0
Nausea/Vomiting	1.4 (0.3)	1.9 (0.2)
Abdominal Pain	1.0 (0.2)	0.7

See PRECAUTIONS, Cough.

Clinical adverse experiences probably or possibly related, or of uncertain relationship to therapy, occurring in 0.5% to 1.0% (except as noted) of the patients treated with ACCUPRIL (with or without concomitant diuretic) in controlled or uncontrolled trials (N = 4397) and less frequent, clinically significant events seen in clinical trials or post-marketing experience (the rarer events are in italics) include (listed by body system):

General: back pain, malaise

Cardiovascular: palpitation, vasodilation, tachycardia, *heart failure, hyperkalemia, myocardial infarction, cerebrovascular accident, hypertensive crisis, angina pectoris, orthostatic hypotension, cardiac rhythm disturbances*

Gastrointestinal: dry mouth or throat, constipation, *gastrointestinal hemorrhage, pancreatitis, abnormal liver function tests*

Nervous/Psychiatric: somnolence, vertigo, syncope, *nervousness, depression*

Integumentary: increased sweating, pruritus, *exfoliative dermatitis, photosensitivity reaction*

Urogenital: *acute renal failure*

Other: amblyopia, pharyngitis, sinusitis, bronchitis, *agranulocytosis, thrombocytopenia*

Fetal/Neonatal Morbidity and Mortality

See WARNINGS, Fetal/Neonatal Morbidity and Mortality.

Angioedema: angioedema has been reported in patients receiving ACCUPRIL (0.1%). Angioedema associated with laryngeal edema may be fatal. If angioedema of the face, extremities, lips, tongue, glottis, and/or larynx occurs, treatment with ACCUPRIL should be discontinued and appropriate therapy instituted immediately. (See WARNINGS.)

Clinical Laboratory Test Findings

Hematology: (See WARNINGS)

Hyperkalemia: (See PRECAUTIONS)

Creatinine and blood urea nitrogen: Increases (T1.25 times the upper limit of normal) in serum creatinine and blood urea nitrogen were observed in 2% and 2%, respectively, of patients treated with ACCUPRIL alone. Increases are more likely to occur in patients receiving concomitant diuretic therapy than in those on ACCUPRIL alone. These increases often remit on continued therapy.

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*In some patients, the antihypertensive effect may diminish toward the end of the once-daily dosing interval. In such patients, an increase in dosage or twice-daily administration may be warranted.

PD-103-MI-7457-C1(052)

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Define The Product Before Trying To Fix It

by

Carol Williams, M.D.
President, Missouri State Medical Association

Carol Williams, M.D.

The country and the states are engaged in a difficult struggle to develop a health care system that works to the benefit of all of its citizens. Almost every segment of the population has a proposal to fix the ailing giant. Organized medicine, the hospitals, business, the elderly, the insurance industry, and the politicians all have pet notions about how to cure it. It will be important for all of the players not to get overwhelmed by the complexity of the problems - to work toward a common vision of what a properly functioning health care system would look like and to devise the system to enflesh that vision.

The first task for those who would develop a workable health care system will be to define the product - what it is that will be the right of every citizen to expect in return for their health care dollar. As a first principle, preventive medicine and public health should be a prime focusing element.

For medicine, this will mean speaking out on those life-style factors that negatively impact the lives of our patients and consume inordinate amounts of health care resources through the diseases generated by these activities. The remedy for these diseases lies not so much with the health care profession as with society and its ability to change its behavior. Medicine will, of necessity, continue to have the responsibility for informing the public; but society itself will have to work on altering its life-style. The health care system developed should include incentives to diminish the demands on its resources for diseases related to such things as smoking, sedentary life-style, poor eating habits, drug abuse, domestic and general violence.

All of us can afford to adopt a preventive medicine way of thinking. Sound immunization programs for all ages. Tracking and contact notification and treatment of persons with communicable diseases must not be victimized by budget constraints. For those with chronic diseases, the proposed system should maximize their ability to remain in the productive stream of life by enhancing their control over their diseases. An annual physical examination with appropriate screening test should be offered as a part of the basic health care package.

To keep perspective in the ongoing discussions about our health care system, the house of medicine will have to continue its advocacy for the patients. It will be imperative that prevention of disease and amelioration of chronic diseases be a major element in the planning. ■

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CRY OF THE HEART

Reflections of a practitioner

By Louis P. Dehner, M.D.

Did you see it? They were only brief vignettes on *60 Minutes* on May 10, 1992, but they said a great deal about the practice of medicine today. We looked into the faces of a roofer, a pet store operator and a young man who left his underserved community; they all have something in common with us since they are all physicians. The only one who still practiced medicine abandoned his attempts at private practice for financial reasons and joined a managed care group, but the other two left medicine altogether since they had been worn to the point of complete despair and frustration with much of medicine as it is practiced today. A fourth physician who is a Trustee of the American Medical Association commented upon the fact that most physicians in this country have reached or are in the process of "burn out."

How are you feeling today, and do you anticipate tomorrow with a sense of dread when you walk into your office, operating room, patient's room, or behind the microscope to diagnose some patient's misery? Are you looking at the patient and concentrating on their medical problem or does that patient represent something else that medicine has been transformed into today? We are not talking about the discharge summary, operative report or laboratory results, but the seemingly endless stack of forms to be filled out for third party payers, quality assurance, workman's compensation and whatever else is placed under our noses to be completed in full or else. We have become like the petty bureaucrats and clerks who control our lives and interfere with our ability to concentrate on the care of patients or as it was formerly referred to - the practice of medicine. We are driven by a clock which is attached to a cash register and together, the essence of the bottom line has been defined. No longer can we carefully work through a patient's diagnostic and therapeutic problems in a fashion that bears any resemblance to a thoughtful, intellectual and careful process. Patients can no longer convalesce in the hospital, but are deposited on the curb side in the accustomed quick, sick fashion. The doctor-patient relationship has evolved into a contentious triangle, rectangle or pentagon of doctor, patient, hospital, third party and regulators in various combinations. Our interactions with each other have also suffered as one physician pushes another in this increasingly contentious, stressful and non-collegial environment.

Why and how is it that we have arrived at this dolorous state of affairs and is there any hope? For our two colleagues in the *60 Minutes* program who abandoned medical practice, their answer to these basic questions was simply to walk away under the assumption that they were unlikely to regain any satisfaction in helping a fellow creature through the trials and tribulations of our human existence. A societal decision has been made that the escalating costs of medical care are largely the consequences of profligate and imprudent actions by thoughtless and selfish physicians whose medical decisions must be taken over by functionaries of government and business; these non-medical types will effectively and efficiently rein in the staggering costs which currently take up about 12% of our gross national product. Let us not forget that the cost of controlling cost itself is driving prices upward and has become the fastest rising component of the health care dollars. It is estimated that one-fourth of the health care dollar is expended for purely administrative purposes. We find ourselves in the current quagmire

Continued on page 334

Heart - Continued from page 333

because medicine and its practice have gradually been taken over by individuals who know nothing about medicine, but are making decisions that affect you and me and our relationships with our patients and each other. It is discouraging and disheartening to find ourselves at the mercy of a high school graduate who is working a computer algorithm that will or will not authorize you to admit or a patient and will determine the time has come for discharge.

As individuals and collectively in our local medical societies and the Missouri State Medical Association, it is more important than ever before for us to stay engaged or to get involved with the activities of these organizations. As individuals we are lost, but it is through the efforts of our medical societies that we can speak and act collectively. Interestingly, the American Medical Association is seeking relief form federal antitrust provisions so that we can bargain as a group with the third party payers (American Medical News, May 11, 1992). Decisions are being made everyday outside our ranks and realm and then presented to us as a fait accompli to either like it or more likely for it to become a lump. The lump has gotten larger for many of us to the point of near or absolute exasperation and a sense of hopelessness. A memorable line from the movie, *Network*, by the late Peter Finch comes to mind when he opened the window and shouted, "I am madder than hell and am not going to take it anymore." Am I the only one who feels this way? ■

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Dear Dr. Woerth,

We completed and returned a survey form about our laboratory testing and personnel that HCFA mailed to us last November. Could you explain why this survey was sent?

M.G.

Dear M.G.,

The survey you received from HCFA is the first step to implement the Clinical Laboratory Improvement Amendment (CLIA) of 1988. The amendment regulates medical laboratory testing, including that done in the physician's office. Completion of the survey form is necessary in order to be certified by HCFA and thus able to bill for laboratory testing performed for Medicare patients. Physicians labs that are not certified or have not received a waiver of certification are unlikely to be reimbursed by Medicare for lab services performed in the office after September 1, 1992.

CLIA rules divide labs into three groups according to complexity of lab tests performed. These three groups are waived, moderate complexity, and high complexity. Medical offices doing only the following test will be eligible for a certificate of waiver.

*Dipstick or tablet reagent urinalysis for bilirubin, glucose, hemoglobin, ketone, leukocytes, nitrate, pH, protein, urobilinogen, specific gravity,

*Ovulation test - visual color comparison determination,

*Erythrocytes sedimentation rate (non-automated),

*Fecal occult blood

*Spun microhematocrit

*Hemoglobin (by copper sulfate),

*Blood glucose (FDA-cleared home use devices).

The cost of the certificate of waiver is approximately $100 and carries with it a stipulation that the medical office agree to follow "good laboratory practice." These labs would not be subject to the inspection, personnel standards, and proficiency testing required for moderate and high complexity labs.

The medical offices providing moderate or high complexity tests will have to pay certification fees of up to $600 and have a director, technical consultant, clinical consultant, and testing personnel. These positions can be filled simultaneously by one person meeting and training and experience qualifications. The training and experience requirements of the director, consultants, and testing personnel are greater for the high complexity labs than for the moderate complexity labs. Both moderate and high complexity labs will be subject to unannounced inspection from HCFA personnel every two years.

HCFA estimates that of physician office labs 5% will be eligible for a certificate of waiver, 85% will be classified as moderate complexity, and 10% will be classified as high complexity.

Dear Dr. Woerth,

I am frequently called in as a consultant for a suspected diagnosis but after examining the patient, I find the suspected disease does not exist. How should I code for this diagnostically?

Dr. R.T.

Dear Dr. R.T.,

First you should code for the symptoms that the patient has and then use ICD-9 code V71.x, observation and evaluation for a suspected condition. You will need to use the appropriate 4th digit (and 5th digit if the patient has a suspected mental condition). Do note that the code had an inclusion note which indicates that the V71 codes are to be used when a patient presents with some symptoms but which after observation and examination shows no need for further treatment or medical care.

Do you have a question for Dr. Woerth? Include your questions along with your name, address and phone number and send them to:

Janice K. Woerth, Ph.D.
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Or call her direct:
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Although your name will not be used in *Missouri Medicine*, please include your name and phone number so that Dr. Woerth may contact you if she needs additional information to answer your question.

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Dr. Donald Sessions, left, served as Editor of *Missouri Medicine* magazine from 1988 through 1991. Dr. Dorothy Reister, 1991-92 MSMA president, presented Dr. Sessions with a plaque in recognition of his service to the Association at the MSMA 1992 Annual meeting in St. Louis, MO.

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ACCEPTANCE SPEECH MSMAA CONVENTION

by Marge Perkins, MSMAA President

When I was membership vice president, I asked how many of you played golf when we met at our four regional meetings. I was using a golf theme for the membership drive. Not very many of you played, but, of course, a lot of your spouses did! However, from visiting your county societies this past year, I think a few more of you really play. But let me tell you, you are all going to know a lot more about golf, especially the terms, by the end of this year as we, the MSMAA, are "Going..To...Go...For(e)....It."

Golf and the Auxiliary have a lot in common. You have to work hard at both before you can see improvements. You can't play golf only once in a while and expect to shoot par, nor can you expect the auxiliary to just keep the meetings interesting and the projects worthwhile. Both activities can be frustrating, aggravating and some "shots" just don't work no matter what you do. But golf and the Auxiliary can be invigorating, challenging and very rewarding when you prepare well and the shots are on the mark.

Golf can be divided into four parts: the drive, the irons, the chip and the putt. The auxiliary can be divided into four parts also: Membership, Health Projects, Legislation, and the American Medical Association Research Foundation (AMA-ERF). We are going to drive long and far for members. We are going to hit crisp irons so that our Health Projects are what our communities need and want. We are going to chip close to those legislators who will vote for the good of medicine and we are going to sink that putt by raising more money than ever from AMA-ERF.

How are we going to do this? In Membership we are "Going To Go For(e) It" by all becoming Membership Chairmen. Our membership is down this year, just as it is nationwide, so if each one of us would get one person to join or rejoin we would double our membership. You have heard this many times but it certainly is true and so easy to do. The District Directors and the Member-at-Large Chairman are going to be working on the counties who had or who have never had an auxiliary to see if they won't join us in this very important organization. Every one is busy, involved in other organizations, working, raising children and so on, but what is more important than belonging to an organization that is supportive of your spouse's work?

We are "Going To Go For(e) It" in Health Projects by carrying on our projects in each county group. One purpose of the MSMAA is to promote better health for everyone. We need to continue state-wide projects and projects you do and support in your communities every year. Or, maybe we need to analyze our communities needs and in coalition with other health groups fill those needs. We need to make our presence felt in the community as this is a vital part of our organization.

We are "Going To Go For(e) It" in Legislation. This is an election year and we need to elect candidates who will vote for the good of medicine, not try to socialize it, especially based upon the Canadian system. Again, the easy things are the best things to do.
1. Register and get your spouse and children of age to register and VOTE! One vote can make a difference.
2. Organize an event to introduce a candidate that you support to your friends and neighbors.
3. Write to your legislators. Tell them how you feel. It can make a big difference when they see hundreds of letters for or against a certain bill.
4. Join MMPAC. As the old saying goes, "put your money where your mouth is." Campaigns are so expensive these days that one more ad might make a difference.

And we are "Going To Go For(e) It" by raising funds for AMA-ERF. Last year $2.4 million was raised nationwide for research and scholarships to medical schools. We certainly want the best minds going to medical school. We will continue the Sharing Card on the state and local level and next year at Tan-Tar-A, there will be a golf buffet with your favorite foods from golf resorts where you can shoot for pars, birdies, or a hole in one, all for the good of medicine.

In closing I want to say that you will be hearing these themes over and over this coming year as we become a team. The Missouri State Medical Association Auxiliary team is "Going To Go For(e) It" by increasing membership, working for better health in our communities, getting involved in the legislative process and raising money for our medical schools. ■

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Ticks- Continued from page 339

tic vials where they were kept alive and returned to our laboratory at MU.

All ticks were identified to species, using light microscopy and appropriate taxonomic keys. Subsequently, five smears were made from each unfed adult as follows: two haemolymph smears were taken by bilateral amputation of tarsus I on each tick and three midgut smears were made following dissection of the abdomen. Ticks were embedded alive into paraffin blocks, covered with sterile brain-heart infusion broth, and the dorsal portion of the abdomen cut away with a no. 11 scalpel, thus exposing the viscera. Fine-pointed tweezers were used to dissect a section of midgut, which was then used to obtain the three remaining smears. Nymphal ticks, being smaller, were mashed singly and the resultant homogenate used to make whole-body smears. Such slides were air dried, acetone fixed, and stored wrapped in foil in a freezer at -70°C until further processing. The remains of ticks thus dissected were stored at a like temperature in individual vials containing brain-heart infusion broth in case additional testing became necessary. Haemolymph smears from adult ticks were tested for *Rickettsia rickettsii* (the causative agent of Rocky Mountain spotted fever) and *Francisella tularensis* (the pathogen responsible for tularemia). The midgut smears from the adult ticks were tested for *B. burgdorferi*, *F. tularensis* and *Ehrlichia* spp. (pathogens in the latter genus may cause ehrlichosis--a rickettsial-like condition). The whole-body smears from the nymphal ticks were tested only for *B. burgdorferi*, and larval ("seed") ticks were too small to process in any manner other than for specific identification.

We processed our tick smears for pathogen identification by using a microimmunofluorescent antibody technique. Briefly, this process employs the immunological ability of antibodies to "recognize" particular antigenic material. Our technique for *B. burgdorferi* and *R. rickettsii* used a direct staining process, in which antibodies formed against the pathogens were conjugated directly with fluorescein isothiocyanate dye. Thus, organisms in smears testing positive ("recognized" by the appropriate antibodies) fluoresced when viewed with a fluorescent microscope. The material we used to test for *Borrelia* was a commercial, polyclonal antibody preparation. While probably capable of recognizing species of

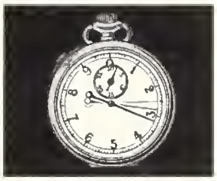
Borrelia other than just *B. burgdorferi*, we felt that it was important not to miss any potential positive ticks. In the event of a positive test, further testing with *B. burgdorferi* monoclonal antibodies could be conducted on the tick remnants stored in the freezer. The antibodies used for *Rickettsia* testing were also directly conjugated with fluorescein dye. Antibodies used to test tick smears for *Francisella tularensis* and *Ehrlichia* spp. were not directly labeled. Here, the fluorescein dye was conjugated with secondary antibody to dog or rabbit antisera specific to *E. canis* and *F. tularensis*, respectively. This required an additional step in the testing process. The anti-*F. tularensis* antibodies were obtained commercially and from Dr. Frances Nano, University of Victoria, British Columbia. Those for *Ehrlichia* were from the Centers for Disease Control, Atlanta, GA. All such antibodies were tested for cross-reactivity to the pathogens under study, with negative results.

In all such laboratory testing, our procedure involved positive controls (smears from known cultures to insure that our methodology was adequate to detect the pathogens). Additionally, the characteristic microscopic morphology of the pathogens provided a secondary check on our accuracy.

Interim results from the 1991 season. The tick species collected during 1991 included the Lone Star, *Amblyomma americanum*; the American dog tick, *Dermacentor variabilis*; the winter tick, *Dermacentor albipictus*; the blacklegged tick, *Ixodes scapularis*; the brown dog tick, *Rhipicephalus sanguineus*; and the Gulf Coast tick, *Amblyomma maculatum*. Of these, the Lone Star and American dog ticks were by far the most abundant and widespread, occurring from northern to southern Missouri. The American dog tick is familiar to most pet owners, being highly associated with dogs, cats, and other domestic animals. The Lone Star tick is likewise widespread in Missouri and well known to cattle producers, outdoor enthusiasts and pet owners. The range of Lone Star ticks has expanded northward in Missouri during the past two decades, following successful wildlife management techniques focused on deer and other game species, which constitute major hosts for the ticks. The larvae ("seed ticks") of Lone Stars occasionally are found on lawns, and cause much consternation when literally thousands crawl up onto unsuspecting people. The brown dog tick and the Gulf

Continued on page 342

US THEM



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Postgraduate Medicine

Ticks- Continued from page 341

Coast tick were collected only occasionally. The latter, a significant pest of cattle in the southern U.S., is a new state record for Missouri. In this study, the winter tick and the blacklegged tick were associated primarily with whitetail deer.

The blacklegged tick, because of its close taxonomic relationship with *Ixodes dammini* (the current name of the species that has thus far been associated with transmission of Lyme disease), was of special interest to us in these collections. During 1991, it was found principally south of the Missouri River, and the adults were strongly associated with whitetail deer. Despite this, the blacklegged tick constituted only a minor portion of the 1991 collections. No specimens classifiable as *I. dammini* were collected.

Preliminary analysis of the 1991 data has indicated that the tick drag technique produced about twice as many tick-positive samples per time interval as did the "probes". With regard to habitat, most tick-positive samples this year were obtained from the forested areas with overhead canopy. This is in contrast to the conventional wisdom that brushy areas harbor the most ticks. A comparison of tick populations between Missouri geographic regions was not made for this single season of collecting.

Some ticks collected during 1991 were positive for the pathogens causing ehrlichiosis, Rocky Mountain spotted fever, and tularemia. About 1.4% of the American dog ticks and 1.6% of the Lone Star ticks collected were positive for *Ehrlichia* sp. These ticks were from locations widespread throughout Missouri. Specific identification of the pathogen is currently being done; however, *E. chaffeensis* has been ruled out.

About 2.7% of the American dog ticks and 6.2% of the Lone Star ticks collected during 1991 tested positive for *R. rickettsii*. These positive ticks were widespread throughout Missouri. In contrast, only 1.8% of American dog ticks were positive for *F. tularensis*, and these all were collected from one region in southwest Missouri.

No ticks collected during 1991 in this study tested positive for *B. burgdorferi*, the etiological agent of Lyme disease. While it is possible that infected ticks might have been missed during our sampling procedure, this seems highly unlikely, particularly if the increasing number of reported Missouri Lyme disease cases represents *bona fide* distribution of the syndrome. Another possibility is that the Missouri situation involves a pathogen other than *B. burgdorferi* and a vector other than *Ixodes* sp. There appear to be significant dissimilarities between the epidemiology of Lyme disease as described from the northeastern U.S. and the syndrome currently affecting Missourians, although the latter seems to be a "Lyme-like" condition. Our intent is to continue with this study for the 1992 season.

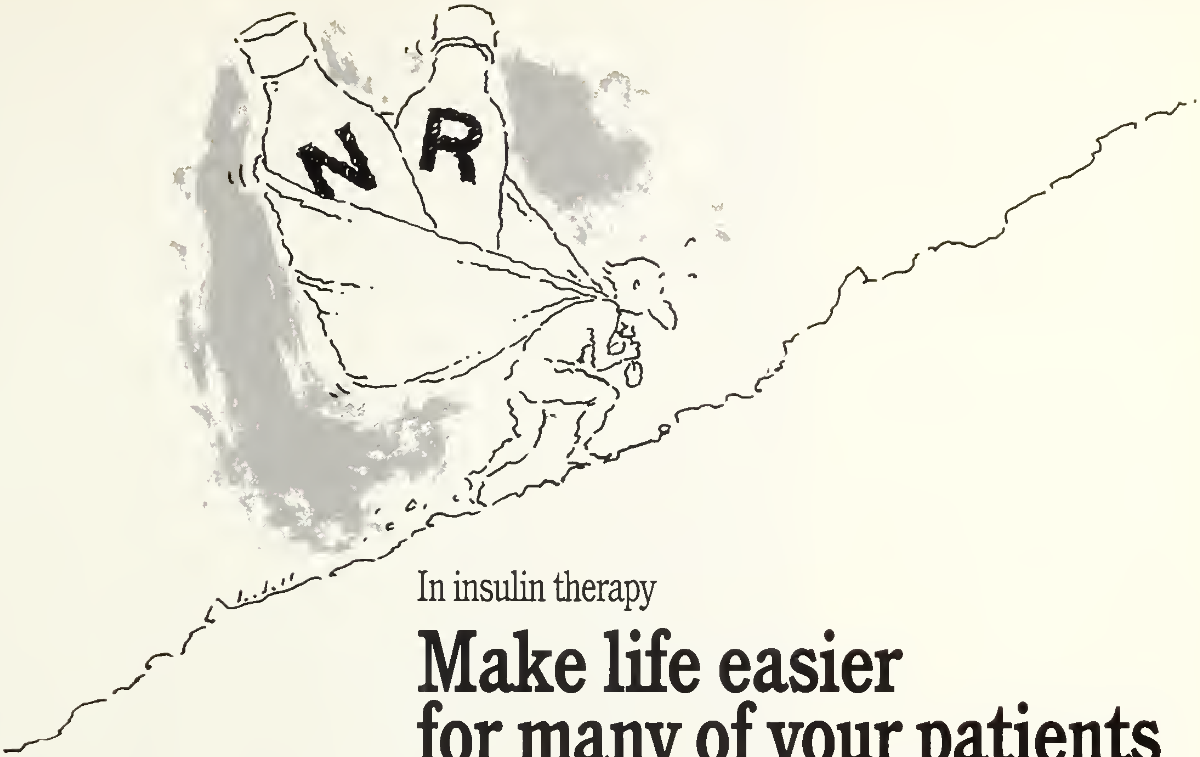
Ticks will remain a fact of life for Missourians, and the possibility of contracting Rocky Mountain spotted fever and tularemia is of genuine concern. While ticks can be avoided by staying indoors, there is no reason to refrain from activities outside if a few common-sense rules are followed. All persons should check for ticks at the end of each day, and carefully remove any found. Prompt removal will markedly decrease the chances of any pathogenic organisms being transmitted. The best way to remove ticks is by using fine-pointed forceps to pull them off slowly. Be careful not to squeeze the internal contents of the tick into the feeding wound. Lastly, avoid leaving the tick mouthparts embedded in the skin. A disinfection with alcohol will complete the job. Ticks can be discouraged by tucking trouser tops into boots, and by wearing long-sleeved shirts while engaged in outdoor activities. Treatment of clothing (not skin) with 0.5% permethrin aerosol (sold commercially as Coulston's (TM) or Permanone (TM)) constitutes the best tick repellent program currently available. Spraying yards with insecticides is generally ineffective as a tick preventive, and is not usually recommended.

The Authors

Dr. Robert D. Hall, K.A. Hewadikaram, and K.E. Doisy are associated with the University of Missouri - Columbia's Department of Entomology. Dr. G.A. McDonald is with the Department of Molecular Micorbiology and Immunology.

For information about this study please contact:

Dr. Robert D. Hall
Department of Entomology
1-87 Agriculture
University of Missouri



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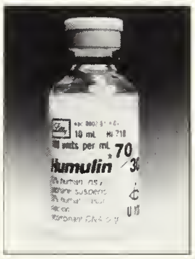

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The University of Missouri School of Medicine At The Turn of The Century

A Blueprint For Reform in Medical Education

Hugh E. Stephenson, Jr., M.D. and Maria L. Evans, M.D.
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With few exceptions, the state of American medical education at the turn of the century was woefully abysmal, and nowhere was this more true than in the state of Missouri. Of the 147 medical schools in existence in 1910, 13 of them were in Missouri--third only to Illinois[18] and New York.[15] Of these, 10 schools were independent, proprietary schools of medicine, "eclectic" schools, or schools of osteopathy, and not affiliated with any university. Most of these schools operated solely for profit, and existed primarily upon student fees.

Although the process of medical education reform had been in effect since the 1880s, and the American Medical Association's Council on Medical Education had been inspecting and ranking the nation's medical schools since 1906, it was Abraham Flexner's report to the Carnegie Foundation in April of 1910, titled "Medical Education in the United States and Canada," that first brought this issue to widespread public attention. Only three schools -- Washington University School of Medicine, St. Louis University School Medicine, and the University of Missouri School of Medicine -- were spared utter humiliation in this report. The University of Missouri medical school had recently scaled down from a four-year school to a two-year "half-school" in 1908. Interestingly enough, it was a pioneer in many of the radical changes that swept through American medical education before and following the report, thanks to the leadership of the university and of the Missouri State Medical Association.

Abraham Flexner

Although much of the credit for American medical education reform has erroneously been directly attributed to Abraham Flexner, his report catalyzed the impetus for reform through mechanisms already framed by the AMA and the various state licensing boards.

The exposure of the proprietary medical schools also exposed the fact that quality medical education was an expensive process. More importantly, the report would serve as another inducement for medical schools to seek university affiliation. Again, the University of Missouri School of Medicine embraced another "radical" idea -- a university affiliated teaching hospital.

As the University of Missouri School of Medicine prepares to move into the 21st century, it becomes even more important to retrospectively explore the role that "the little old two-year school in McAlester Hall" played in shaping the future of medical education over 90 years ago.

National and State Perspectives in Medical Education

Even before the Carnegie Foundation's report, the problem of unifying and standardizing medical education had been addressed at both the national and state level of organized medicine. The Council on Medical Education (CME) of the American Medical Association was formed in 1904. At its first annual meeting, the Council developed "minimum" and "ideal" standards.

The minimum standard consisted of

1. Preliminary education sufficient for admission to any freshman class of a recognized university;
2. The passing on said credentials by a state official;
3. Graduation from an approved medical college requiring four years' course work of not less than 30 weeks per year with 30 hours of work per week of actual work; and
4. Passing of an examination for licensure before a state board.

The ideal standard consisted of

1. An accredited four-year high school education;
2. One year university training in physics, chemistry, and biology;
3. A four-year medical school course (as outlined above); and
4. One year of post-graduate medical internship.[1]

Most Missouri medical schools would have had great difficulty meeting even the minimum standard.

In 1907 the CME of the AMA reported on a detailed, planned series of medical school inspections similar to the one undertaken by the Carnegie Foundation two years later.[2]

The 1908 conference compared and contrasted medical education in the United States versus that of the 20 other important nations of the world. This study revealed that a five-year medical course (four years of medical school followed by a year of internship) appeared to be the "world standard" at that time.[3]

The CME's 1909 conference outlined a recommended curriculum -- what is now considered the "classic" medical school curriculum -- consisting of two years of basic science instruction followed by two years of clinical clerkships in the major medical disciplines.[4] The CME's report was actually the basis for the "model curriculum" outlined in the Flexner report.

The Missouri State Medical Association's role in improving medical education pre-dates even the CME of the AMA. As early as 1878, the MSMA appointed a Committee on Medical Education to investigate the facilities of Missouri medical schools. Of the 12 schools then in existence, only four were declared representative of "legitimate" medicine in Missouri. In 1879, this same committee proposed the adoption of a plan requiring all medical graduates in the state to hold a college degree in "arts, sciences, and languages," a truly progressive notion for the times.[5] Indeed, by 1909, the leaders of organized medicine in Missouri were well aware of the changes

University of Missouri Medical School students (1907) dissect cadavers as part of their training. (University of Missouri Archives photo.)

needed to shed the state's odious reputation as a haven for proprietary medical schools and their associated quackery. As stated in 1910 by the MSMA CME President, Dr. Tinsley Brown of Hamilton, Missouri, "It is evident from a study of the medical schools in this country and their work that there are five specially rotten spots which are responsible for most of the bad medical instruction. They are Illinois, Missouri, Maryland, Kentucky, and Tennessee." He further said, "...the principal cause for this bad work is found in the existence of medical schools which are conducted solely for profit."[6]

Other reforms initiated through the MSMA that sounded the death knell for the proprietary medical schools included the development of city/county medical societies, standardization of state board exams, and the development of the Missouri State Board of Health. Another major factor was the state's emerging influence on licensure. Prior to 1901, Missouri's medical schools had the authority to grant medical licenses. The Hall Bill of 1901 took this privilege from the medical schools and gave it to the State Board of Health.

The University's Perspective

First established in 1841 as an affiliate of Kemper College, and later recognized in 1872, the University of Missouri Medical School enjoys the title of "the oldest medical school west of the Mississippi." The medical school began with a two-year course in 1873, increasing the curriculum to a three-year and four-year course in 1891 and 1892, respectively.[7]

In 1907, Dr. Pinkney French of St. Louis, made a $300,000 donation of the tangible and real property belonging to Barnes Medical College in St. Louis. The Board of Curators decided to transfer the final two years of the medical school to St. Louis, beginning with the 1908-1909 school year. However, the donors failed to clear $90,000 of indebtedness incurred on the property, so the University was never able to receive the gift.[8] (In retrospect, it is interesting to note that the Barnes Medical College had recently lost and subsequently regained its accreditation. One can only speculate Dr. French's "hidden agenda.") Despite the fact that the Columbia campus had its own hospital -- Parker Hospital, a 45-bed facility opened in 1902 -- it was generally felt by both the Board of Curators and President Richard H. Jesse that the University could not support a high-quality four-year program. This spurred the return to a two-year "half-school" in 1908. University medical students would have to seek their final two years of training elsewhere. In addition, the Board of Curators anticipated Flexner's visit, and feared he would find the program's clinical instruction deficient. The decision did not, however, affect the students already enrolled, and the classes of 1909 and 1910 completed their education at Columbia.

The decision to downsize the medical school was

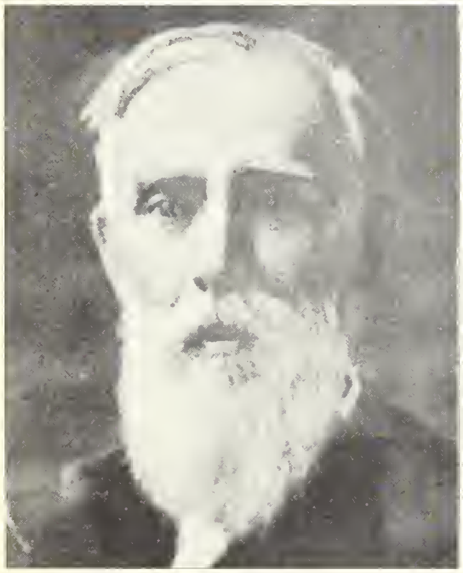
Parker Memorial Hospital and Medical Building. (University of Missouri Archives Photo.)

fairly unpopular among alumni and practicing physicians, but President Jesse stood firm on his decision. "What I have said hitherto will be disputed by no intelligent man or woman; but here -- on the usual way to teaching clinics -- comes one bone of my contention. Almost everywhere it is assumed that if the hospital and dispensary have patients enough, the teachers be numerous enough and famous enough as practitioners, and the time 18 months, the instruction will be excellent, provided, of course that it is based on 18 months spent previously in the laboratories aforesaid. But here -- on this assumption -- let me enter my protest and plead for radical revision of the methods prevailing everywhere, or nearly everywhere, in America."[9]

The Carnegie Foundation's Perspective

Although the CME of the AMA had been reporting annually for five years, and its first inspection of medical schools was completed as early as 1907, its members felt that a report from outside the AMA could exert more pressure for reform in medical education. Many council members already expressed concern that resentment of the AMA over its efforts toward reform was already growing steadily. The CME and its chairman, Arthur Dean Bevan, believed that the opinion of an independent, disinterested expert would carry more weight than the AMA's own recommendations. Bevan asked Carnegie Foundation to undertake the task, as the CME believed that the foundation's prestige would considerably speed the closure of the nation's proprietary medical schools. Its president, Henry S. Pritchett, a Missourian, commissioned secondary school educator Abraham Flexner. His report, "Medical Education in the U.S. and Canada," was published in 1910. Pritchett was well aware that the Carnegie Foundation report underscored the original work of the AMA, as well as the AMA's desire to downplay its involvement, as a November 4, 1909, letter to Bevan illustrates: "In all this work of the examination of medical schools, we have been hand-in-glove with you and your committee. In fact, we have only taken up the matter and gone on with the examination very much as you were doing, except that as an independent agency disconnected from actual practice, we may do certain things which you perhaps may not. When our report comes out, it is going to be ammunition in your hands. It is desirable therefore, to maintain in the meantime a position which does not animate an immediate connection between our two efforts."[10] The CME also agreed to delay publishing its list of "satisfactory" schools until the appearance of the Carnegie Foundation's report, in order to add more weight to its own report. In fact, the AMA was so successful in keeping a low profile that 80 years later, it is Flexner and the Carnegie Foundation that are erroneously credited with reforming medical education, instead of the AMA.

It is important to note that Pritchett's choice of Abraham Flexner to author this report was not without its shortcomings. Flexner was neither physician nor scientist (his background was in education administration and he was well-versed in the classics), so he had no first-hand knowledge of the scientific method. Medical science, medical history, medical sociology, and contemporary systems of health care delivery were all foreign to him. He did, however, possess amazing energy, drive, and a knack for exposé writing. The result was a report that covered several superficial aspects of American medical education, outlined an ideal curriculum, discussed financial aspects, outlined a model basis for state board exams,


Andrew McAlester

Richard Jesse

and championed postgraduate medical education. Although in retrospect the report appears somewhat superficial, Flexner managed to describe conditions at every medical college in the United States and Canada concisely. He also included his commentary on medical education for women and African-Americans.

Abraham Flexner had little use for hyperbole and "whitewashing," hence his report is filled with delightfully vivid (albeit graphic) descriptions of the conditions in American medical schools at the turn of the century. He describes the anatomy collection of one California medical college as "a small box of slides and the dried-up filthy fragments of a single cadaver."[11] He reported that an "indescribably filthy" anatomy laboratory of a Kansas school "was simultaneously used as a chicken yard."[12] An osteopathic school in Philadelphia boasted that their students were allowed to study at "every important surgical clinic in the great medical colleges and hospitals of that city"; Flexner revealed that these students could gain access to those hospitals only by concealing their identity.[13]

Flexner's travels brought him to Missouri twice, in April and November 1909. He appeared to be quite impressed with the University medical school during his Columbia visit in April 1909. "The medical department occupies a new and well-equipped building, excellently adapted to its purposes," he writes. "The teaching is in charge of full time instructors of modern training and ideas. A university hospital of 45 beds gives the department the advantage of clinical material and connections, even though the actual instruction is limited to the work of the first two years, a feature of great importance."[14] Flexner was also impressed that the leadership at the University "had the wisdom and courage to confine its efforts to the first two years."[15] He also pointed out that the University was one of only 22 medical schools in the country that demanded two years of college before admission, beginning in the 1910 school year -- a full year more than the AMA's "ideal standard." Thus, Missouri joined the ranks of schools such as Johns Hopkins, Harvard, Rush Medical College, and Western Reserve in that regard.[16]

In contrast, the report painted a rather sordid picture of medical education in several other Missouri schools. "Professors" at the American School of Osteopathy in Kirksville were, in reality, senior students.[17] The St. Louis College of Physicians and Surgeons owned physiology laboratory equipment, but it was never used, and a make-believe display laboratory sat, unused, on tables gathering dust.[18] With the exception of Washington University, St. Louis University, and the University of Missouri, the report called the medical schools in Missouri "utterly wretched."[19]

A tremendous drop in the number of medical schools ensued over the next three decades. By 1922, only 81 medical schools remained, and 30 years later, (1950) that number had plummeted to 65. Not until the 1960s would this country see an increase in the number of medical schools.

By downsizing and redefining its mission, the University of Missouri Medical School managed not only to survive the Carnegie Foundation's report, but actually to become a leader in requiring higher standards of its matriculants. When the four-year curriculum was restored in 1956, the school was ready to meet the demands required of the two clinical training years, moving into its new facility, the University of Missouri Medical Center (now the present University Hospital and Clinics.

Although many contributed to Mizzou's successful 1909 inspections, the lion's share of the credit goes to "the father of the University of Missouri School of Medicine," Dr. Andrew W. McAlester. Born, fittingly enough, in 1841 (the same year as the University School of Medicine), Dr. McAlester has a "track record" that includes a long and illustrious history of medical reforms in Missouri. During the post-Civil War era, he reorganized the MSMA and helped unify the voices of "northern" and "southern" sympathizing physicians for statewide health care reform. In 1901, he helped spur the state legislature into taking action against proprietary medical colleges and combatting quackery through higher licensing standards.[20]

In 1903, McAlester addressed the MSMA on the subject of higher standards in medical education. "The present trend of affairs clearly indicates that the profession as a whole desires to establish and maintain a still higher standard of excellence," he stated. "A thoroughly scientific medical education rests upon such a broad foundation that a greater familiarity with collateral sciences is necessary that is the case with any other profession."[21] As the School of Medicine's dean from 1880 to 1909, he undoubtedly spearheaded many of the school's reforms prior to the Carnegie Foundation's report.

This year, 103 fourth-year medical students walked across the stage in Jesse Hall Auditorium to receive their

M.D. degrees. Perhaps it is fitting that the medical school holds its commencement in the building named for President Richard Jesse. Had it not been for his foresight, along with that of Andrew McAlester and the Board of Curators from 1900 to 1910, the "little old two-year school in McAlester Hall" might not have escaped the scrutiny of the Carnegie Foundation, and thus allowed to become the quality institution it is today. ■

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Isolated Traumatic Accessory Splenic Rupture: A Case Report and Literature Review

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The spleen is the most commonly injured organ in blunt abdominal trauma. Overall mortality from ruptured spleen is as high as 19%, due to the frequency of associated injuries.[1] Many conditions are associated with spontaneous splenic rupture, which may render the spleen relatively more vulnerable to blunt trauma. Conditions contributing to spontaneous rupture include infectious mononucleosis, tumors of the spleen, leukemia, malaria, typhoid fever, subacute bacterial endocarditis and pregnancy.[2] Because an accessory spleen may reflect a pathologic process much as the parent spleen, it too may be increasingly susceptible to blunt injury. In 1966, Huebner and Reservitz reported a case of traumatic rupture of both a spleen and its accessory spleen in the setting of mononucleosis; both splenectomy and accessory splenectomy were required. The patient in this case was a first-baseman, struck by the base runner during a softball game.[3]

An accessory spleen, or splenculus, is believed to be present in 10% of the population.[2,4,5] Isolated injury to an accessory spleen is relatively rare, perhaps due to its relatively small size and somewhat protected intra-abdominal location.

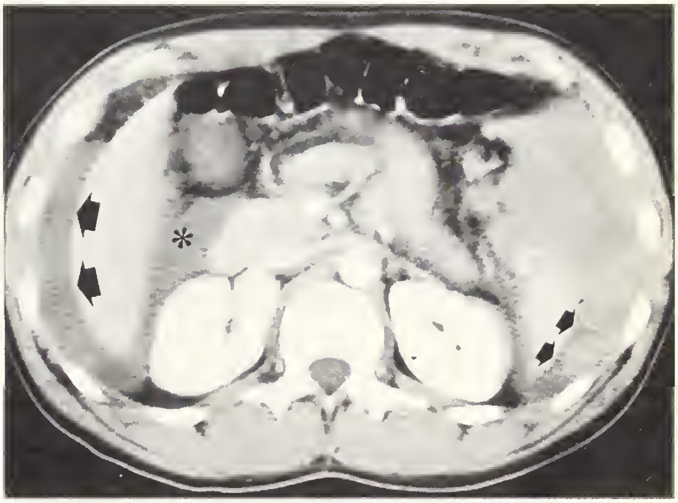
Case Report

A 32-year-old white male state penitentiary inmate was struck in the left upper abdomen while running to first base during a softball game in the prison recreation area. He underwent a brief examination in the infirmary, but no further studies were necessary until the following day, when he complained of continued left upper quadrant pain and nausea. The patient was noted to have orthostatic hypotension and tachycardia, which resolved after intravenous administration of crystalloids. A CT scan of the abdomen was interpreted as revealing a splenic fracture with free intraperitoneal hemorrhage.

The patient was transferred to the University of Missouri Hospital. He had continued abdominal pain, some left lower chest pain, and some left shoulder pain.

Upon physical examination, the patient was noted to be a slender well developed white man in mild distress. Blood pressure was 116/64, pulse 90, respirations 20, temperature 37.2° C. The abdomen had a well healed irregular scar in the left upper quadrant from a previous superficial laceration. There were hypoactive bowel sounds and the abdomen was diffusely tender with rebound,

FIGURE 1



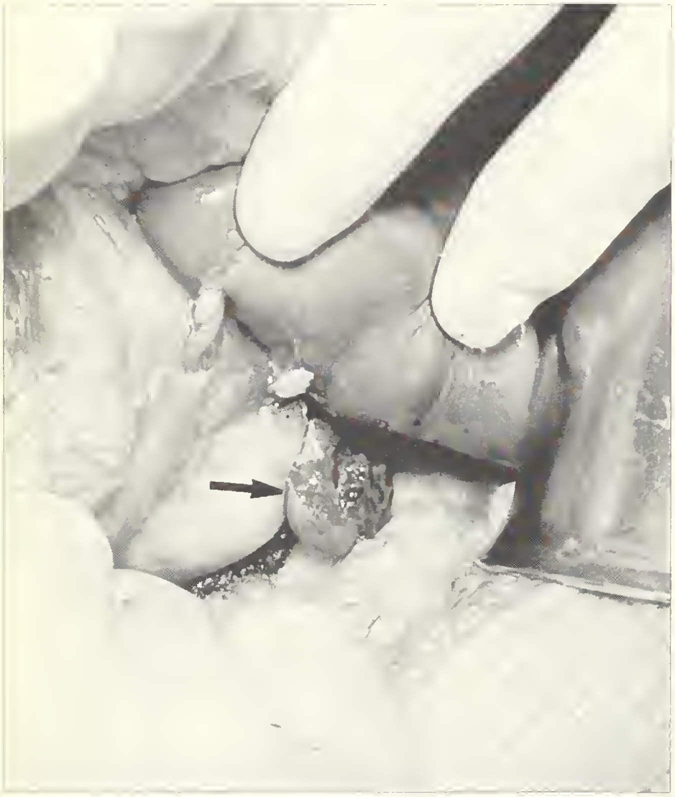
CT scan of the abdomen caudad to the splenic hilum. Note the free intraperitoneal hemorrhage at arrow.

most marked in the left upper quadrant. The left lower side of the chest was tender along the costal margin anteriorly.

Admission laboratory values included a hemoglobin of 9.2 grams with a hematocrit of 25.3%. White blood cell count was 15,100. Coagulation studies gave normal results. Chest radiograph revealed neither rib fractures nor other abnormalities. The previously done CT scan of the abdomen was reviewed and free intraperitoneal fluid was noted.

Pneumococcal vaccine and additional intravenous fluids were administered in the emergency department. The patient was taken to the operating room where, through a midline laparotomy, more than two liters of clotted blood was evacuated from the peritoneal cavity. Examination of the spleen revealed no rupture; however, a nearly avulsed 1.5 cm accessory spleen was discovered actively bleeding near the lower pole of the splenic hilum. Accessory splenectomy with ligation of supply vessels controlled the bleeding. The spleen, normal in size, remained intact. Transfusion of two units of packed cells was required perioperatively. The postoperative course was unremarkable, with the hematocrit remaining stable at 28%. The patient was discharged on four days postoperatively. Microscopic examination of the accessory spleen revealed no abnormalities.

FIGURE 2A



Intraoperative photogrpahy of accessory spleen (arrow) near the lower pole of the splenic hilum.

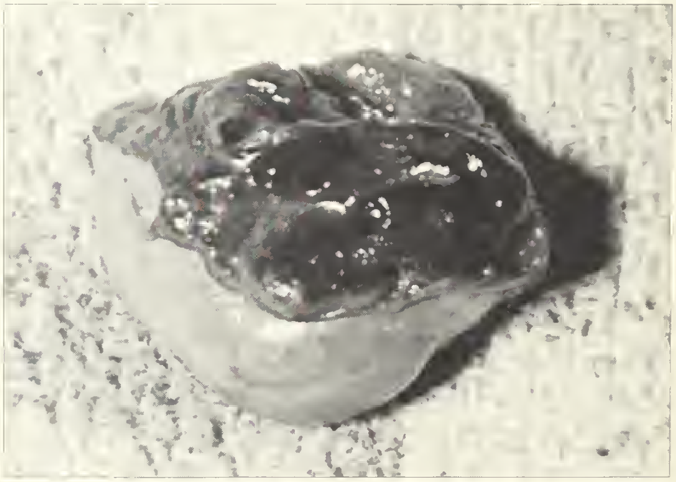
Discussion

We found only two other reported cases of isolated rupture of an accessory spleen. In 1962, Kumar reported a traumatically ruptured accessory spleen discovered during an appendectomy. The patient had fallen in the bathroom pre-operatively and injured the lower left side of his chest. Splenectomy and accessory splenectomy were required in this 8-year-old boy when a large hematoma developed near the splenic hilum.[6]

A second case believed to have resulted from minimal trauma, if any, was reported by Texeira and Hardin in 1974. An 11-year-old Mexican girl developed abdominal symptoms after horseback riding. A left upper quadrant mass was palpable and plain x-ray films suggested a 9 cm mass was displacing the splenic flexure of the colon. Intravenous pyelography confirmed no renal abnormalities. Laparotomy revealed hemorrhage from a 2 cm accessory spleen into a cystic cavity. The cyst was not lined with epithelium, and was believed to be a walled off area of hemorrhage rather than a congenital cyst. The accessory spleen was without pathologic abnormality and the patient recovered uneventfully.[7]

Most studies have recorded the incidence of an accessory spleen in 10% to 15%[5,8] of people. Some earlier studies have suggested as many as 44% of patients undergoing

FIGURE 2B



Surgical specimen of isolated traumatic accessory splenic rupture.

exploratory laparotomy for hematologic diseases[9] have an accessory spleen. Accessory spleens are single in approximately 90% of patients, although as many as 25 coexisting splenculi have been reported.[8,10] When there are more than one, accessory spleens are in the same location in 85% of cases. Accessory spleens are most commonly about 1 cm in diameter, although they range from 0.2 cm to 6 cm.[8] A splenculus is usually located near the splenic hilum or pedicle (75%) although a significant percentage (1 in 6) have been found in the tail of the pancreas on autopsy.[8] Less commonly, a splenculus is found on the greater omentum along the greater curvature of the stomach, along the splenic artery, in the wall of the small bowel, or the mesentery of the large or small bowel. Rarely, the splenculus may be associated with the left ovary or left testicle due to the splenic-gonadal fusion during the early embryologic period.[2,5,8,10]

The gross and microscopic structure of the accessory spleen is essentially the same as that of a parent spleen. Both have a well formed capsule and fibrous trabecula. These features distinguish splenculi from splenic implants, or splenosis, which occur in the peritoneal cavity after splenic rupture. In splenosis, the nodule capsules and trabeculae lack muscle and elastic tissue. Furthermore, the implants lack malpighian follicles with a central arteriole.[2,5]

Summary

Although small and relatively protected, an accessory spleen can be the cause of acute intraabdominal symptoms following minimal trauma. Although a splenic fracture or laceration may not be apparent on by CT scan, the scan cannot rule out the possibility. The diagnosis of splenic injury should be entertained in light of appropriate history and physical findings. The case we present and prior similar cases are a useful review of isolated accessory splenic rupture due to blunt trauma.

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Kayaking As A Risk Factor For Leptospirosis

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Leptospirosis is a common zoonosis present throughout Missouri. Previously regarded as an occupational illness of farmers and sewer workers, it is now primarily acquired from exposure to water polluted with the bacteria Leptospira. We present a case report of leptospirosis acquired through kayaking and review both the disease and the risk factor associated with kayaking.

Leptospirosis, considered the most common zoonosis in the world, is caused by the spirochete *Leptospira*. Its presence in Missouri is well documented.[1] Its natural reservoir includes both domestic and wild animals. Humans become infected when abraded skin or mucous membranes are exposed to the urine of an infected animal either directly or through contaminated water. Although previously considered an occupational illness of farmers and sewer workers, studies reveal that it occurs in individuals of all walks of life[2,3] as well as in all age groups.[4]

Despite the bacteria's ubiquitous presence, the disease remains underdiagnosed.[2] In Missouri there have been only 16 cases reported over the past six years.[5] With the increased use of Missouri streams and rivers there will doubtless be more exposure to this bacteria and subsequently more cases. Of particular concern is the sport of kayaking. While any exposure to polluted water can result in contracting this illness,[6-9] kayakers prefer high water and are more frequently in contact with the water, making kayaking a recognizable risk factor. We present a case report and review the factors that make kayaking an exposure risk.

Case Report

On March 23, 1991, a 37-year-old male physician was kayaking the St. Francis River in Madison County, Missouri, during the Missouri Whitewater Championships. The river that day was at high water and the previous day had been in flood stage. The patient suffered no injuries or unusual occurrences but as part of normal kayaking had his head and hands submerged several times in the water. Twelve days later he had the sudden onset of fever, diffuse myalgias and headache. Over the next three days he had daily fevers to 103-104°F, chills, anorexia and generalized weakness. On the fifth day of his illness he was hospitalized.

The patient had not traveled outside the United States recently and had not been exposed to cattle or other livestock. He had no recent exposure to meningitis, hepatitis or toxins. He had received no skin abrasions or accidents while on the river. He reported no recent tick bites. This trip was his first kayaking of the year and he denied any other exposure to contaminated water.

Physical examination revealed an ill appearing white man in no acute distress with a temperature of 102.8°F, blood pressure 128/70, pulse 88/min, and respiratory rate of 20/min. The sclerae were mildly icteric, mucous membranes were markedly dry. The neck was supple without adenopathy; the lungs were clear, the cardiac examination revealed normal rate and rhythm. Abdominal examination showed no hepatosplenomegaly, no tenderness or masses. The neurological examination was unremarkable. There were no rashes.

Laboratory values were WBC 12,200 with 56% neutrophils, 31% band forms, 5% lymphocytes with 8% atypical lymphocytes, hemoglobin 14.8 gm/dl, hematocrit 42.4%, platelet count 218,000. A chemistry profile showed glucose 127 mg/dl (normal 70-110), sodium 133 mmol/l (normal 140-148), potassium 3.9 mmol/l (normal 3.6-5.2), chloride 94 mmol/l (normal 100-108), total CO_2 25.9 mmol/l (normal 21.0-32.0), urea nitrogen 16 mg/dl (normal 7-18), creatinine 1.2 mg/dl (normal 0.6-1.3), LDH 271 U/L (normal 100 to 190), creatine kinase 77 U/L (normal 21-232), SGOT (AST) 84 U/L (normal 15-37), SGPT (ALT) 95 U/L (normal 30-65), alkaline phosphatase 171 U/L (normal 50-136), total bilirubin 4.5 mg/dl (normal 0.0-3.0), calcium 8.8 mg/dl (normal 8.8-10.5), albumin 3.6 g/dl (normal 3.4-5.0), uric acid 3.3 mg/dl (normal 2.6-7.2), cholesterol 83 mg/dl (normal 0-200), triglyceride 69 mg/dl (normal 30-200). The urine qualitatively contained bilirubin, ketones, protein and blood. Microscopically it had 0-3 WBC's and 0-3 RBC's. Febrile agglutinins and cold agglutinins were negative and serological testing for hepatitis A and B were negative. An ultrasound of the abdomen revealed a contracted gallbladder but was otherwise normal. Chest films were negative.

Because of the possibility of both rickettsial disease as well as leptospirosis, the patient was started on intravenous doxycycline and amoxicillin. Blood cultures were negative as were initial titers for antibodies to rickettsial illnesses and leptospirosis. Within 48 hours the patient became afebrile. His bilirubin began to return to normal but his liver enzymes continued to rise and peaked on the eighth day of illness with LDH 374 U/L, SGOT (AST) 701 U/L, Gamma GT 124 U/L, and alkaline phosphatase 175 U/L. Headaches which had initially resolved with abatement of the fever returned on the eighth day of the illness and were associated with lethargy. This resolved the following day and no spinal tap was performed. No further fever developed and the patient was sent home on oral doxycycline. Weekly titers were performed for rickettsial illness and leptospirosis. On the fourth week after the onset of illness, leptospirosis titers rose to 1 to 800.

Discussion

This case demonstrates the multisystem presentation of leptospirosis with the typical protean non-specific symptoms. While Weil's disease with its renal failure and poorer prognosis did not develop, there were still significant liver abnormalities. The abrupt onset after an incubation period of about 10 days was typical (range 2-26 days).[10] The most common symptoms which then follow are severe headache, gastrointestinal symptoms, and myalgias. A characteristic finding at this time is a conjunctival suffusion which develops in 33% of cases.[9] Neutrophilia, elevations in serum transaminase values, anemia, and hyponatremia may be present.[11] Following a one to three day period of improvement with decrease in temperature, there is a second stage with up to 95% of cases displaying conjunctival suffusion, myalgias, nausea, vomiting and abdominal tenderness.[12] An aseptic meningitis develops in 50% of patients during this time with abnormal CSF in up to 90% of cases.[12] Leptospira are not isolated from the spinal fluid during this phase; the inflammatory response is believed to be caused by immune complexes.[13] Our case demonstrated the development of the "second stage" with the worsening of headaches and change in mental status. The lack of conjunctival suffusion was the only "classic" feature that failed to develop (seen in 33-85% of patients).[13]

Weil's syndrome is a particularly severe form of the disease and comprises 5% to 10% of recognized cases. Jaundice appears between day 2 and 9 and may continue for several weeks. During the second phase, renal and hepatic manifestations dominate. Acute tubular necrosis occurs with increased blood urea nitrogen and serum creatinine. Marked serum hyperbilirubinemia develops. Thrombocytopenia occurs in up to 50% of cases.[12] Mortality usually results from renal failure rather than hepatic failure and is less than 10% in the United States.

While leptospirosis usually follows a benign course with spontaneous recovery, it is important to establish the correct diagnosis. If antibiotic therapy is started within the first four days of illness, the duration of fever and the risk of complications can be reduced.[14] Penicillin, cephalosporin, and erythromycin have been reported effective,[15] but the drug of choice is doxycycline in a dose of 100 mg twice a day.[14] Children under the age of eight can be treated with parenteral penicillin G 200,000-250,000 units/kg/day.[16]

Due to the non-specific symptoms and the lack of physical findings in this illness, it is not surprising that many of the less severe forms go undiagnosed.[9] Yet leptospirosis was first recognized as a distinct clinical entity in 1886 by Weil.[17] In 1915 Indada established the etiology of Weil's disease[18] and within a few years, the rat was recognized as a major host factor throughout the world.[19] Later Leptospiral etiologies were established for a variety of endemic or epidemic diseases previously recognized as distinct entities. These included autumnal fever and seven-day fever in Japan, canefield fever in Australia and swamp or mud fever and swineherd's disease in Europe.[3] The association of Leptospiras with disease in dogs was reported as early as 1937.[20] Leptospirosis became known as an occupational disease of farmers and those exposed to rats (hence the term "sewer worker's disease").

Yet one of the most frequently associated risk factors for leptospirosis was not the exposure to livestock or rats per se, but rather the exposure to water contaminated by the infected animals. As early as 1941, a small outbreak of seven cases of "Weil's disease" in bathers was reported by Havens.[21] The epidemic possibilities of this route were demonstrated in the United States when Schaeffer reported 50 cases of leptospirosis in a group of 80 people swimming in creek located near a livestock pasture.[22] In 1975 Tennessee reported a cluster of seven cases of children who apparently acquired the infection while swimming in a stream.[23] Oregon reported five cases in loggers whose only likely source of infection was their exposure to surface water.[2] In one of the largest studies of leptospirosis in the United States,[3] 19% of 485 cases reviewed involved immersion, largely through swimming in stagnant water. In this same study it was found that occupation was no longer a major risk factor and was largely incidental to contracting this illness.

Records from the Missouri Department of Health show that over the past six years, water exposure was a factor in six of the seven cases where a clinical history was available. Two of the cases were kayakers. Interestingly, the only Missouri fatality during this time was an occupational exposure in a farmer.[5]

Kayaking as a sport seems to be particularly suited for contracting this illness. While canoeing involves some exposure to water immersion, particularly in the summer when it may be accompanied by swimming, it does not usually involve the intentional pursuit of high water that is found in kayaking nor the repeated immersion of the face and head in the water.

Most kayakers seek water levels that are not suitable for recreational canoeing. These higher waters are more likely to contain run off from barn yards, dumps and animal burrows than normal flows and thus are more prone to contain Leptospira. In addition, when such levels are encountered, kayakers will then "play" the water. Activities such as "enders" in which the boat is pulled underwater by a forceful current to a depth that both the boat and its paddler are then hurled into the air, frequently result in the boater finding himself or herself upside down in a strong current. While the skilled paddler will quickly "roll" the boat right side up, head and hands will have been exposed to the water. A single day on a river may result in multiple such exposures.

Other pursuits include "surfing" and "hole-riding." Both activities involve using the hydraulics of falling water to hold a boat in place in the midst of a rapid. At such time the swift flowing water is usually splashed forcefully against the kayaker's face, hands and chest. And owing to a kayaker's low position in the water, even flat water paddling exposes the hands more frequently to water and increases the risk of contact with Leptospira. The two previous Missouri cases of leptospirosis in kayakers had the common factors of boating in flooded waters and immersion in the water.[24]

Furthermore, the popularity of these sports continues to grow. The three largest kayak clubs in the state report continued gains in membership. The Missouri Whitewater Championships on the St. Francis River attract boaters from across the country and entrants have grown in number each year. In addition, recent information from outfitters that sponsor international kayaking have revealed that leptospirosis is an increasing problem from other countries and information regarding this illness is now routinely given to their international travel clients.[25]

Conclusion

Leptospirosis is an underdiagnosed illness which can infect individuals of all age groups and all walks of life. It's protean and non-specific symptoms make it difficult to diagnosis and it is frequently not included in the differential diagnosis of the febrile patient. Exposure to polluted water and recreational kayaking would seem to be particular risk factors and should alert the physician to the possibility of this disease. ■

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Acknowledgment:
The author wishes to acknowledge the assistance of the Missouri Department of Health and particularly Dr. F.T. Satalowich and his staff in providing information regarding Leptospirosis in Missouri.

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Aggressive Rehabilitation Following Anterior Cruciate Reconstruction: An Update and Protocol

By Martin M. Pomphrey, Jr., M.D.
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Dramatic changes in anterior cruciate ligament (ACL) reconstruction surgery and rehabilitation methods have occurred during the past few years. These developments are reviewed briefly, and a rapid rehabilitation protocol is presented which facilitates the safe return of the patient to his or her pre-operative functional level in sports and activities of daily living.

The past decade has seen a dramatic and continuing evolution in the rehabilitation of knees following anterior cruciate ligament (ACL) reconstruction. In the early 1980s most ACL reconstruction was being done as an open procedure, and the knee was immobilized in flexion, using a cast for up to six or more weeks before initiating rehabilitation. Patients were kept non-weight bearing on crutches during that period of time. The methods of graft fixation, as well as the graft tissue itself were felt to be weak in the early post-operative period.[1]

A multitude of problems arose from this rather conservative approach.[2,3,4] These included thigh (quad) atrophy, patellofemoral pain and chondromalacia, failure to regain full extension, and slow return of function to the involved limb. This paper will review certain aspects of ACL reconstruction and rehabilitation[3,5,6] A rapid rehabilitation protocol designed to prevent such complications and to hasten a patient's safe return to sports and other recreational pursuits in presented.

It is, of course, imperative that a complete history, adequate physical exam, and proper diagnostic tests be performed to confirm an ACL injury. Only then can the patient be given proper counseling regarding treatment options and be prepared for reconstructive surgery should that option be chosen. The timing of surgery is critical. It is not uncommon to hear of patients having ACL reconstruction within a few days of injury. Recent reports indicate a much lower incidence or arthrofibrosis and postoperative knee flexion contracture in those patients whose surgery was delayed for three weeks or more following their injury[7,8] The delay of surgery allows the patients to begin rehabilitation, restore knee range and motion, muscle control, eliminate swelling, and prepare themselves both mentally and physically.[6]

Technical ability has made great strides since those early days of ACL reconstruction. Most ACL surgery is now performed arthroscopically. The use of central third patella tendon/bone autograft is the gold standard, but other graft tissue is showing comparable results.[9-14] The graft can be secured with interference screws,[15] introduced endoscopically, which provide fixation strong enough to allow immediate range of motion and weight bearing. In addition, research has demonstrated that the graft tissue at the time of surgery is stronger than the original ACL.[16] Assuming isometric graft placement at surgery, the graft will not fail with early aggressive rehabilitation.

Closed kinetic chain exercise principles are important[3,7,8,17,18,] especially in the early rehabilitation after ACL surgery. These exercises require the foot to be fixed in a weight bearing position and make the knee inherently more stable. This type of exercise is thought by some to aid tri-contraction of the quadriceps, hamstrings, and gastrocnemius muscles. These muscles are dynamic knee stabilizers, which in effect cause compression of the menisci and joint surfaces when they contract, thereby eliminating the amount of possible anterior tibial displacement and increasing joint congruency. By doing so, unnecessary stresses are eliminated from the ACL. Henning et al[19] found over 50% more ACL elongation during active resistive knee extension exercises (open chain) than during a squat, an example of closed chain exercises.

The following in an outline of a clinically successful protocol for rehabilitation following ACL reconstruction. The basic tenets have been used for seven years with a failure rate of less than 1%.

This rehabilitation protocol is offered as a guide. Individual patient needs, availability of certain pieces of equipment, personal bias of physicians, therapists, and trainers may require adjustments or changes.

Experience indicates this protocol is safe and is effective in returning patients to their desired level of activity.

I emphasize the importance of maintaining knee extension early in the rehab process. Once lost, it is most difficult to regain. Formerly patients were placed in a Bledsoe brace at surgery with hinges set to allow 0-60 degrees range of motion. Virtually all patients rested with the knee in flexion. At two weeks post-op, patients lacked an average of 7.5 degrees of terminal extension.

The patients are now immediately placed in a knee immobilizer or a hinged knee brace locked in full extension. They keep the knee fully extended for the first two weeks post-op, except for time spent on the CPM machine or doing rehab exercise. Since making this change in early bracing, patients have improved extension at two weeks post-op. The average range of motion at two weeks for the last twenty-five cases has been negative 2.7 to 91 degrees. These results are similar to those reported in Barrett's study.[5] This is a significant improvement, especially when considering the difficulty in regaining lost extension during rehabilitation. ■

Phase 1

1. **Day of Surgery:**
    - a) **CPM machine 0-45 degrees up to 6-8 hours as tolerated.**
    - b) **Knee immobilizer with knee in full extension when not using CPM.**
2. **First Day Post-op:**
    - a) **CPM 0-60 degrees up to 6-8 hours per day.**
    - b) **Every 2 hours prop foot on pillows and allow knee to straighten out full. Maintain for 10 minutes.**
    - c) **Walk on crutches wearing knee immobilizer, weight bearing as tolerated.**
    - d) **Continue intermittent use of ice on knee.**
    - e) **Begin active range of motion of knee out of brace 45-90 degrees.**
    - f) **Isometric quads/hamstrings co-contractions at 0 degrees, 30 degrees, 60 degrees, and 90 degrees.**
    - g) **Begin straight leg raises in all 4 directions.**
    - h) **Ankle pumps.**
    - i) **Gluteal sets.**
    - j) **Instruct patient in prone hangs with 1-2 pound ankle weight.**
    - k) **begin electrical stimulation to quads 4-6 hours per day.**
    - l) **discharge home if patient is comfortable.**
3. **Post-op Days 2-5:**
    - a) **CPM at home 1 hour at least 4 times a day. Add 5-10 degrees of flexion per day, as tolerated.**
    - b) **Prop foot on pillow or towel every 2 hours to maintain extension.**
    - c) **Prone hangs with weights q.i.d. times 10 minutes.**
    - d) **Increase walking as tolerated.**
    - e) **Electrical stimulation, quad sets, straight leg raises, PT exercises as instructed in hospital.**
    - f) **Use EGS as needed to decrease swelling.**
      SWELLING = SPEEDOMETER
4. **Post-op Days 5-7:**
    - a) **First office visit.**
    - b) **Measure for functional brace.**
    - c) **Continue knee immobilizer. May wean crutches, as tolerated at home.**
    - d) **Discontinue CPM if adequate motion has been obtained.**
    - e) **Begin Air-dyne in physical therapy. Use moveable handle bars to help regain knee motion.**
    - f) **Begin patella mobilization at therapy and perform at home q.i.d.**
    - g) **Continue prone hangs.**
    - h) **Begin calf raises, one-quarter deep knee bends (mini-squats), lateral step-ups 4-6".**
    - i) **Continue electrical stimulation.**

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PHASE II

5. Two Weeks Post-op:
    - a) Suture removal.
    - b) Pick up functional knee brace and discontinue knee immobilizer.
    - c) Discontinue electrical stimulation, if making adequate progress.
    - d) Start Stairmaster in PT.
    - e) Begin leg curls on Eagle, Nautilus, or Isokinetic machine from 90 degrees through 30 degrees.
    - f) Begin leg press machine from 90-45 degrees using both legs.
    - g) Terminal knee extension with 1-5 pound ankle weight or comparable Theraband.
    - h) May walk/run in pool with wet vest or swim if available.
    - i) Graduate to conventional exercycle at low resisance.
6. Four Weeks Post-op:
    - a) Start skater slide (if available) with brace on.
    - b) Start knee extension with Eagle, Nautilus, or Isokinetic machine, concentric only, 90-40 degrees.
7. Two Months Post-op:
    - a) Add Nordic Track, if available.
    - b) Continue above and increase as tolerated. Increase weights and endurance as tolerated.
    - c) Increase exercycle endurance to at least 20 minutes.
8. Three Months Post-op:
    - a) Exercycle resistance moderate.
    - b) Increase exercycle to 30 minutes or more.
    - c) Start eccentric quads on Eagle, Nautilus, or Isokinetic machine 30-90 degrees.
    - d) Increase concentric quads.
    - e) Isokinetic evaluation with 30 degree stop at 180 and 240 degrees per second.

PHASE III

9. Four Months Post-op:
    - a) Isokinetic evaluation with 30 degree stop at 80 and 240 degrees per second. If 70 percent of normal knee, may start jogging with brace, lateral shuffles, cariocas, jumping rope.
    - b) Continue weights and other rehab activities to regain strength.
    - c) Increase resistance on exercycle or Lifecycle.
10. Six-Nine Months Post-op:
    - a) Isokinetic evaluation at 60, 180, and 240 degrees per second.
    - b) Consideration for return to athletic competition if at least 90 percent of strength has been regained.

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FEATURE ARTICLE

The Missouri Cardiovascular Health Plan: A Focus on Prevention

By Sue Dabney, M.Ed, M.P.H., Carol Smith, M.S.P.H., Ross Brownson, Ph.D., Anne Goldberg, M.D. and Tom LaFontaine, Ph.D.

The authors describe a framework for planning, implementing, and evaluating cardiovascular health programs in Missouri. Populations targeted for intervention are identified, and the role of the physician in supporting community-based cardiovascular health promotion is explored.

CVD in the United States and Missouri

Cardiovascular disease (CVD) is the leading cause of death, disability, and health care expenditures in the United States. Almost one in two Americans dies of CVD, and more than one in four has some form of the disease.[1] Persons with CVD incur over one-third of the nation's total estimated health care costs for adults each year.[2]

Cardiovascular disease mortality rates in Missouri mirror those of the United States. In 1989, CVD accounted for 22,304 deaths in Missouri, representing 45% of all deaths. This is almost twice the mortality rate of cancer, the second leading cause of Missouri deaths.[3]

In Missouri, death rates for most types of CVD declined from 1950 to 1989. Since the late 1960s, there has been a dramatic decline in CVD deaths due largely to a decrease in coronary heart disease (CHD) mortality.[3] Although improved medical care is responsible for some of the decline, it appears that life-style changes, including reduction in CVD risk factors, account for much of the decrease in heart disease mortality.[4-6]

Declines in CVD risk factors since the 1960s are well documented.[4-6] Smoking among American adults has declined. Control of high blood pressure has improved. Americans are consuming less cholesterol, less saturated fat, and less total fat. Blood cholesterol levels have dropped. People also seem more aware of the benefits of physical activity.

Despite these overall improvements in CVD mortal-

Missouri
Cardiovascular
Health
Task Force

The Missouri Cardiovascular Health Plan:
A focus on prevention

CVD in the US and Missouri
- Scope and Trends

High Risk and Priority Populations
- Blacks
- Women
- Lower Socioeconomic Status
- Children

CVD Risk Factors
- Cigarette Smoking
- High Blood Cholesterol
- Overweight
- Stress
- High Blood Pressure
- Physical Inactivity
- Diabetes

Community-based Health Promotion

Goals and Objectives for the Year 2000
- Risk Reduction
- Surveillance and Data
- Services and Protection

Table 1. Content of the Missouri Cardiovascular Health Plan

ity rates, there is still a high percentage of Missourians who have one or more of the modifiable CVD risk factors; 27% currently smoke, 17% have high blood pressure, 20% have high blood cholesterol, 58% are physically inactive, and 29% are overweight.[7]

Missouri Cardiovascular Health Task Force

To meet this health care challenge, the Missouri Department of Health, with funding from the centers for Disease Control, created the Missouri Cardiovascular Health Task Force. The task force, which first convened in August 1990, is comprised of 27 concerned physicians, nurses, health educators, and other health professionals from across the state.

The mission of the task force is to assist and advise the Missouri Department of Health in coordinating, focusing, and expanding CVD prevention and control activities in Missouri. Specific responsibilities include: 1) establishing prevention priorities for Missouri; 2) recommending target populations for intervention and education; 3) identifying providers and mechanisms to implement education, intervention, and control programs; and 4) educating policy makers on the importance of legislation on clean indoor air and the restriction of tobacco sales to minors.

The Missouri Cardiovascular Health Plan

In 1991 the task force developed a framework for planning, implementing, and evaluating cardiovascular health programs and policies in Missouri. It has been released as a 35-page document, The Missouri Cardiovascular Health Plan, which includes background information on CVD risk factors, identifies high risk and priority populations, and establishes statewide goals and objectives for cardiovascular health. (see table 1). The plan targets physicians, public health professionals, legislators, community leaders, and educators.

Target Population

The task force recommended that four segments of Missouri's population be targeted for CVD intervention. These populations - Blacks, persons of lower socioeconomic status, women, and children - were identified as being at higher risk for CVD or as not receiving adequate cardiovascular health education and resources.

Blacks

Date from the 1990 census show that Blacks constitute nearly 11% of Missouri's population and comprise almost 82% of the minority population.[3] Disparities exist in CVD risk factor prevalence between Blacks and Whites (see figure 1). The severity of high blood pressure is greater among Blacks.[1] Obesity is the most prevalent nutrition-related problem for the Black community, particularly women. Data indicate that Black women are almost twice as likely to be overweight as White women.[8] Blacks are less physically active than Whites.[9] Adult Black men have higher smoking rates than White men, although a lower percentage of Black men are heavy smokers.[10] Diabetes-related mortality rates for Blacks in Missouri are roughly twice the rates for Whites, and about two-thirds of deaths in people with diabetes are caused by CVD.[3,11] These issues have been discussed in more detail in an earlier Missouri Medicine article.[12]

Persons of Lower Socioeconomic Status

Socioeconomic status is usually defined by one or more of the following variables: income, education, and occupation.[13] Rates of CVD are higher among lower socioeconomic status populations (see figure 2).[14] Although health experts have not identified all the reasons for this relationship, higher prevalence rates of uncontrolled high blood pressure, smoking, physical inactivity, and obesity are found in the lower socioeconomic status population.[13]

Women

Heart disease is the number one killer of Missouri women, killing nearly 11,720 women in 1989.[3] One in nine American women between 45 and 64 years of age has some form of CVD, and the ratio climbs to one in three at age 65 and beyond.[1] As women age they appear to lose their protection against heart disease. Women over 50 years of age are more likely to develop high blood pressure than men their age.[15] Women over 65 years are less physically active than younger women, and in all age groups, women are less active then men.[16,17]

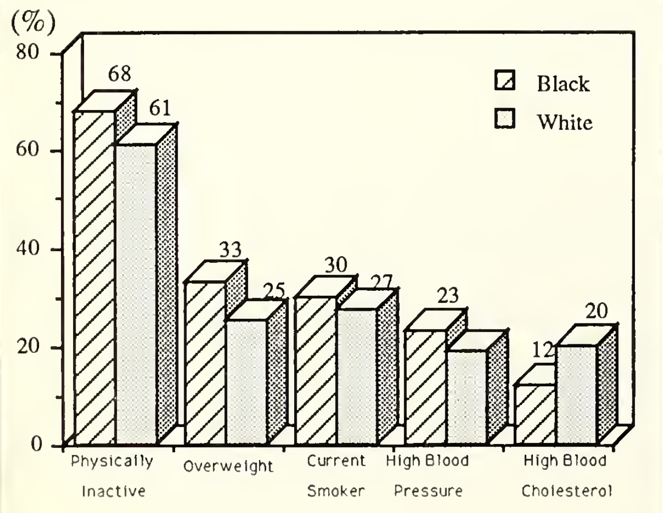
A 55-year-old woman who smokes and has high blood cholesterol is one third more likely to have a heart attack than a man with the same risk factor.[18] The use of oral contraceptives increases a women's risk of heart disease. This risk increases with age and the simultaneous presence of other risk factors, especially smoking.[19] In addition, women who have heart attacks are twice as likely as men to die within the first few weeks.[20]

Children

Studies from several states show that between 28% and 33% of children have at least one elevated CVD risk factor.[21] Although American children are more active than adults, it is estimated that 34% of 10 to 17-year-old children are not participating in physical activity that improves cardiovascular health.[22] The rate of obesity in children ages 6-11 has increased 54% since the 1960s, while the rate in children ages 12-17 has increased 39%.[23] Tobacco use is also a significant risk. Chewing tobacco, smoking cigarettes, or living with parents who smoke may lower children's high density lipoprotein cholesterol and increase their risk of coronary heart disease.[24] A recent study of tobacco use patterns among Missouri adolescents showed that 30% of girls and 28% of boys in the 12th grade had smoked during the previous week.[25]

Community-Based Health Promotion

Figure 1



The task force identified community-based health promotion programs as a necessary approach to modifying CVD risk factors and reinforcing heart healthy behaviors. Changing habits, such as eating, exercising, and smoking may begin at the individual or family level, but maintaining change relies on community reinforcement and approval. A community that promotes exercise activities, a worksite that maintains an employee fitness program, and health care providers that encourage personal responsibility for wellness give individuals the best chance to maintain a heart healthy life-style. Typical community-based health activities include walking clubs, nutrition demonstrations, and smoking cessation classes.

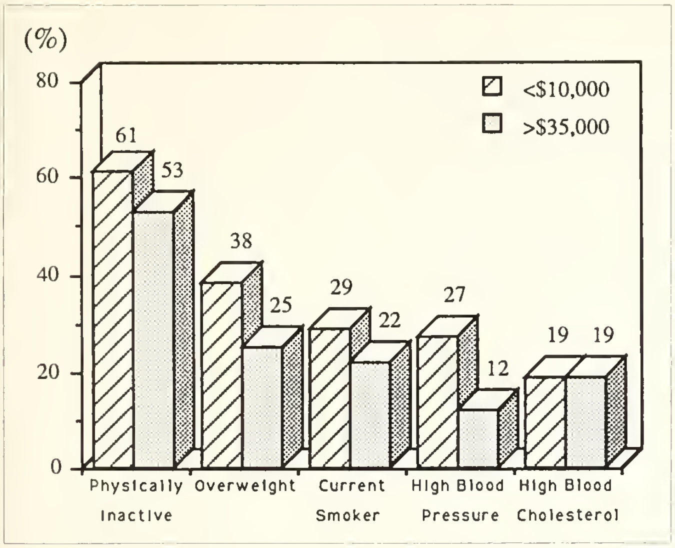
Physician Involvement

Any community-based health promotion program will be more successful with the full support of the medical community. Most people see a physician at least once a year.[26] Therefore, physicians can influence the level of cardiovascular disease prevention practiced in their community. In a clinical setting physicians can 1) Screen patients for CVD; 2) identify and educate patients on CVD risk factors and heart healthy behavior; 3) relate these risk behaviors to the social influences causing or enhancing them; 4) seek a firm, personal commitment from the patient to change behavior; 5) suggest reasonable behavioral goals and offer alternatives to reach them; 6) defuse patient rationalizations and defenses for not changing; and 7) encourage, motivate, and inspire the patient to change.[27] To overcome constraints on physician time and resources, patients can be referred to nurses, dieticians, health educators, exercise physiologists, and community programs for follow-up, education, and skills training for specific behavior change. With proper advice, support, and education, many patients can effectively manage diseases such as abnormal lipids, hypertension, and diabetes without pharmacological intervention.

Working within the community model, physicians can give advice and support to the community-based program coordinators. Physicians are often seen as role models in the community. Physician involvement in these community activities, and support for patient participation in these activities, lend credibility to such initiatives.

In 1980, the Missouri Department of Health surveyed 296 Missouri primary care physicians (general practice, family practice, internal medicine, and obstetrics-gyne-

Figure 2



cology).[28] The survey included questions on current activity levels in CVD prevention and early detection. Results showed that 81% of respondents measure the blood pressure of their adult patients at each visit, 97% always or usually take a smoking history, and 66% always or usually check the serum cholesterol of their adult patients.

These physician practices involving CVD prevention and early detection play a significant role in implementing community-based health promotion programs. Therefore, the Missouri Cardiovascular Health Plan includes objectives directly aimed at physicians. These objectives, based on the national and Missouri year 2000 objectives, consist of increasing the proportion of primary care physicians who report a high or very high level of 1) tobacco cessation education or counseling; 2) nutrition education or counseling; 3) physical activity education and counseling; 4) ordering serum cholesterol for adult patients as part of an initial checkup; and 5) measuring the blood pressure of adult patients as part of each office visit.

The task force believes that with help from physicians, public health professionals, policy makers, educators, and most of all individuals and communities, the decline in CVD mortality in Missouri can be enhanced.

For a copy of the Missouri Cardiovascular Health Plan, write to: Sue Dabney, Missouri Department of Health, 201 Business Loop 70 West, Columbia, MO 65203; or call 314-876-3200.

Acknowledgements

Centers for Disease Control - project funding and technical advice from Dick Lasko, Senior Evaluation Specialist.

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201 Business Loop 70 West
Columbia, MO 65203

WHEN THE BLOOM IS OFF THE ROSE

-- What to do When the Residency of your Dreams isn't

By David Springer, M.D., and Maria L. Evans, M.D.

We've all experienced broken romances and probably know how to deal with them. However, what about the romance of residency? What happens when Match Day is over and you realize at some point in your residency that your specialty choice is not what you want to do, or your program is not where you want to be? Most new physicians have spent several years planning the career of their dreams, and if that career is not what they presently want, the disillusionment, denial, and mental anguish can be devastating. With proper planning, the mid-residency career change can be less stressful.

Step 1:

Do not just decide what you want to do -- decide what you **don't** want to do.

Make a list of the things you like about the practice of medicine. Then make a list of the things you absolutely **don't** like about the practice of medicine. Think about what specialties best fit your list. Likewise, if you like your present specialty, but not your location, make lists of what you like about your present program and what you don't like. Also, take a good look at the city in which you live. Often, "I don't like this program" translates to "I don't like this **town.**" Refer to these lists often, and use them as the absolute guide to your next move.

Step 2:

Use the telephone! Call your friends in different cities and different specialties. Find out how satisfied they are with their programs. (Obviously, few residents are ecstatic about their programs, but many are satisfied.)

Call your practicing physician friends. Find out about the job market in various fields. Learn from them what the "bread and butter" of the job entails. (For instance, you may find pediatric endocrinology fascinating, but you may not find working with several brittle diabetics per day quite as fascinating!) Find out if the demand for the specialty will meet your expectations for the type of locale in which you wish to practice.

Call the programs of interest. Find out how competitive the field (and job market) is. Find out what options are available. Will you have to repeat your R-1 year? Will they help you with moving expenses? One can't make too few phone calls during this process.

Step 3:

To match or not to match? Unlike your experiences as a fourth-year medical student, you will have many more options regarding the match. Many programs may wish to hire you outside the match; some may insist that you go through the match. Decide what works best for you.

Step 4:

If you're still not sure, a transitional year or a year of internal medicine never hurts. Many specialties require prerequisite months in general internal medicine or transitional year. A year of general medicine opens many options in many fields -- and a successful evaluation from such a program carries more weight in a competitive specialty when being evaluated in an applicant pool heavy in fourth-year medical students.

Step 5:

Don't be afraid to "shoot the moon." The worst that can happen is that the program of your dreams says "no." You **can** find love the "second time around!"

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■ Kenneth I. Bluestone, M.D., 1001 E. 101st Terrace, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Bluestone received his medical degree at the University of Missouri, Kansas City, MO, in 1987. He completed his residency at Barnes Hospital, St. Louis, MO. Dr. Bluestone specializes in anesthesiology.

■ Jeffrey A. Bredemann, M.D., 1001 E. 101st Terrace, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Bredemann received his medical degree at the University of Missouri, Columbia, MO, in 1987. He completed his residency at the University of Missouri. Dr. Bredemann specializes in anesthesiology.

■ Paul R. Callegari, M.D., 3635 Vista Ave. at Grand Blvd, P.O. Box 15250, St. Louis, MO 63110-0250, has become a member of the Missouri State Medical Association. Dr. Callegari received his medical degree at the University of Toronto, Toronto, Ontario, Canada, in 1983. He completed his residency at the University of Alberta, Edmonton, Alberta, Canada. Dr. Callegari specializes in plastic surgery.

■ Clifford M. Gall, M.D., 4320 Wornall Rd, Suite 328, Kansas City, MO 64111, has become a memberof the Metropolitan Medical Society of Greater Kansas City. Dr. Gall received his medical degree at Harvard Medical School, Boston, MA, in 1985. He completed his residency at Brigham & Women's Hospital, Boston, MA. Dr. Gall specializes in neurosurgery.

■ Lehman W. Godwin, M.D., 3443 S. National, Springfield, MO 65807, has become a member of the Greene County Medical Society. Dr. Godwin received his medical degree at St. Louis University, in 1988. He completed his residency at the University of Kansas Medical Center, Kansas City, KS. Dr. Godwin specializes in internal medicine.

■ Willie E. Lawrence, Jr., M.D., 4949 Rockhill Rd., Suite 2132, Kansas City, MO 64110, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Lawrence received his medical degree at Harvard Medical School, Boston, MA, in 1984. He completed his fellowship at The John Hopkins Hospital, Baltimore, MD. Dr. Lawrence specializes in cardiology.

■ Creig M. Lobdell, M.D., 1900 S. National, Suite 1800, Springfield, MO 65804, has become a member of the Greene County Medical Society. Dr. Lobdell received his medical degree at the Mayo Medical School, Rochester, MN, in 1987. He completed his residency at the Mayo Graduate School of Medicine, Rochester, MN. Dr. Lobdell specializes in anesthesiology.

■ J. Mark Matthews, M.D., 1001 E. 101st Terrace, Kansas City, MO 64131, has become a member of the Metropolitan Medical Society of Greater Kansas City. Dr. Matthews received his medical degree at Bowman Gray School of Medicine, Salem, NC, in 1983. He completed his residency at St. Lukes Hospital, Kansas City, MO. Dr. Matthews specializes in anesthesiology.

■ Daniel R. Mrosak, M.D., 1220 Franklin, P.O. Box 37, Lexington, MO 64067, has become a member of the Lafayette-Ray County Medical Society. Dr. Mrosak received his medical degree at the University of Minnesota, Minneapolis, MN, in 1982. He completed his residency at Truman Medical Center, Kansas City, MO. Dr. Mrosak specializes in general surgery.

■ Cynthia K. Powell, M.D., 301 West Liberty, Farmington, MO 63640, has become a member of the Mineral Area Medical Society. Dr. Powell received her medical degree at the University of Oklahoma College of Medicine, Oklahoma City, OK, in 1984. She completed her residency at Oklahoma Children's Hospital, Oklahoma City, OK. Dr. Powell specializes in pediatrics.

■ Kenneth D. Stone, M.D., 1012 North Main Street, Sikeston, MO 63801, has become a member of the Southeast Missouri Medical Society. Dr. Stone received his medical degree at McMaster University, Hamilton, Ontario, Canada, in 1988. He completed his residency at MacMaster University Medical Center. Dr. Stone specializes in family medicine.

■ Robert C. Strunk, M.D., 400 S. Kingshighway, St. Louis, MO 63110, has become a member of the Missouri State Medical Association. Dr. Strunk received his medical degree at Northwestern University Medical School, Chicago, IL, in 1968. He completed his residency at Cincinnati Children's Hospital. Dr. Strunk specializes in allergy, immunology and pediatrics.

Continued on page 370

NEW MEMBERS

Continued from page 369

■ Armin J. Wagman, M.D., 1000 E. Primrose #350, Springfield, MO 65807, has become a member of the Greene County Medical Society. Dr. Wagman received his medical degree at the University of Michigan Medical School, Ann Arbor, MI, in 1980. He completed his fellowship at the University of Iowa Hospitals and Clinics. Dr. Wagman specializes in pediatric cardiology.

Residents

Jeffrey W. Colyer, M.D., *Plastic Surgery*
Steven M. Kory, M.D., *Psychiatry*
Marcus Nashelsky, M.D., *Pathology*
Manzoor A. Tariq, M.D., *Internal Medicine*
Charles Yieng-Chu Su, M.D., *Internal Medicine*

Students

St. Louis University
William C. Horn
Sean A. McEnaney
Fred W. Rottnek

University of Missouri-Kansas City
David Bennett
Anurekha Bongu
Kaladevi Chandramohan
Jennifer A. Elliott
Anthony R. Justesen
Alexander Karmazin
Priya Krishna
Sunil Madan
Sri Reddy
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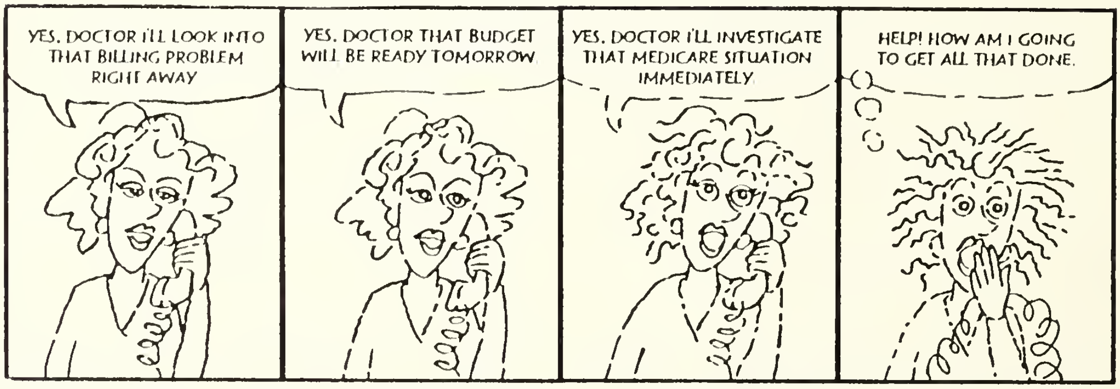
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Sheila Shah, M.D., St. Joesph

Cape Girardeau
Richard L. Kies, M.D., Cape Girardeau
Michael C. Trueblood, M.D., Cape Girardeau

Clay-Platte
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Doctor's Name ____________________

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City/State/ZIP ____________________

Office Phone (To be used only if added info is needed)
(   ) ____________________

Current Medical Specialty(ies) ____________________

Former Medical Specialty(ies) (for past 10 years) ____________________

List surgical procedures routinely performed ____________________

State(s) where currently licensed ____________________

States where formerly licensed (for past 10 years) ____________________

Amount of Professional Liability Insurance now carried
$ ____________________

Is the doctor involved with delivery of babies? ☐ YES ☐ NO

02

■ **Arnoldo Kuczer,** M.D, St. Louis, died April 29, 1992, at age 62. Dr. Kuczer received his medical degree at Facultad de Ciencias Medicina de la Universidad de Buenos Aires, Buenos Aires, Argentina, in 1955. He was a member of the American Academy of Ophthalmology and Otolaryngology and the American Physicians Fellowship, Inc.

■ **Dale F. Burton,** M.D., Hannibal, MO, died April 8, 1992, at age 52. Dr. Burton received his medical degree at the University of Illinois Medical School, Chicago, IL, in 1963. He had served as a major in the U.S. Air Force and was chief of surgery and medical director at Beale Air Force Base, CA. Dr. Burton joined the Hannibal Clinic in 1970 as a physician surgeon and served as chief of surgery at several local hospitals. He was a member of the Tri-County Medical Society, the American College of Surgeons, the American Abdominal Surgeons, the Hodgen Traveling Surgical Club, and the Missouri State Surgical Society.

■ **Joseph L. Johnston,** M.D., Springfield, MO, died April 4, 1992, at age 86. Dr. Johnston received his medical degree at Kansas University Medical School in 1930. He graduated with the highest honors in his class, being accepted to the Alpha Omega Alpha Honor Medical Society. He began practicing obstetrics and gynecology in Springfield in 1936. During his career he had delivered 11,170 babies. Dr. Johnston was a member of the Central Association of Obstetricians and Gynecologists, the American College of Obstetricians and Gynecologists, and the American College of Surgeons.

■ **Florence Q. MacInnis**, M.D., Kansas City, Missouri, died April 17, 1992, at age 87. Dr. MacInnis received her medical degree at Marquette University School of Medicine, Milwaukee, WI, in 1928. She was director of tuberculosis control in Milwaukee and joined the Wisconsin Anti-Tuberculosis Association as a consultant in the 1940s and traveled and lectured through Europe and the Far East. She was a staff physician at several Kansas City hospitals and was a consultant to the Kansas City Health Department for more than 30 years. Dr. MacInnis was a former staff president at St. Mary's Hospital, past director of the Heart of America Tuberculosis Association, past president of the Mississippi Valley Conference on Tuberculosis and the Missouri Thoracic Society. She was also a member of the American College of Chest Physicians and American Society of Internal Medicine.

■ **Thomas G. Otto**, M.D., Cape Girardeau, Missouri, died April 9, 1992, at age 73. Dr. Otto received his medical degree at the University of Alberta School of Medicine, Edmonton, Canada, in 1943. He served in the Royal Canadian Army Medical Corps in Canada, England and Northwest Europe. Dr. Otto arrived in Cape Girardeau in 1950 during a polio epidemic and formed a clinic to provide outpatient services. He developed the Crippled Children's Clinic. Dr. Otto was also a staff member at Southeast Missouri Hospital and St. Francis Medical Center. He was a fellow of American College of Surgeons, appointed Examiner for the American Board of Orthopaedic Surgery, a member of the Cape Girardeau Medical Society, American Medical Association, Mid-Central States Orthopaedic Society, St. Louis Orthopaedic Society and American Academy of Orthopaedic Surgeons.

■ **Sigmund Tashma,** M.D., Clayton, Missouri, died April 20, 1992, at age 98. Dr. Tashma received his medical degree at Loyola University Stritch School of Medicine, Maywood, IL, in 1918. His specialty was urological surgery. He was a member of the St. Louis Metropolitan Medical Society.

■ **Laura Walters,** M.D., Columbia, Missouri, died March 30, 1992, at age 80. Dr. Walters received her medical degree at the Universitaet Wien, Medizinische Fakultaet, Vienna, Austria, in 1937. She was a dermatologist.

■ **Doris C. Gilpin,** M.D., St. Louis, Missouri, died April 29, 1992, at age 68. Dr. Gilpin received her medical degree at Pritzker School of Medicine of University of Chicago, Chicago, IL, in 1948. She was a member of the American Psychiatric Association and the Academy of Child Psychiatry.

UNIVERSITY OF MISSOURI - COLUMBIA

MU medical students launched a new program to educate the public about the benefits of organ and tissue donation. The Gift of a Life program (GOAL) intends to dispel myths about donating organs, to inform the public how to be donors, and to show how recipients and their families benefit from such gifts.

Philipp Othmer is co-director of GOAL at MU and also serves as national coordinator of the project for the American Medical Student Association. He will present information about the program, June 16-20, to the Association of Organ Pocurement Organizations in Washington D.C.

A major objective for the program is to encourage people to tell their families the decisions they would want made if they were involved in an accident. GOAL will take its message to schools and community groups. To schedule GOAL speakers call Philipp Othmer at 874-7813 or Greg Schneider at 442-3209.

Medical students at the University of Missouri-Columbia collect and box used textbooks for shipment to health care workers in Sierra Leone. (Photo by Marguerite Keeley)

For the second year faculty and students packed up used medical textbooks destined for African health-care workers. More than 40 boxes filled with 400 textbooks are destined for Sierra Leone, a small nation on the west coast of Africa.

Air Force Captain **Phil Jackson,** a graduate of the MU School of Medicine, coordinated the project. While he was participating in Project Medflag, a program in which Air Force and Army personnel set up clinics in Third World nations, he learned of the need for more current medical knowledge. He discussed the need with **Ted Groshong, M.D.,** the school's associate dean for student affairs, while on leave in Columbia. As a result, students collected books during the fall 1990 semester and shipped them to Germany. The books were then distributed in Guinea-Bissau, another country on Africa's west coast.

This year medical students took a pickup load of books to Scott Air Base near St. Louis. The Air Force transported the books to Germany. A Medflag team distributed them in Africa in May.

Susan Buckelew, Ph.D., associate professor of physical medicine and rehabilitation, recently received the James Garrett Early Career Achievement Award from the American Psycological Association. The award recognizes the production of a substantial body of work in a relatively short period of time.

Robert W. McMurray, M.D., a postdoctoral fellow in immunology and rhematology, recently presented a paper titled "Prolactin and Murine Systemic Lupus Erythematosus" at the third International Conference on Systemic Lupus Erythematosus in London. He also presented two posters, one of them co-authored by **Sara E. Walker, M.D.,** professor of immunology and rheumatology.

Brent M. Parker, M.D., professor emeritus of cardiology, received an Alumni Achievement Award May 9 from his alma mater, Washington University School of Medicine.

The MU Medical Alumni Organization honored 10 individuals, including MSMA President **Dorothy Reister, M.D.,** during a special luncheon at the MSMA annual meeting in St. Louis. The winners included six alumni of the school and four individuals selected as honorary alumni.

Harold L. Kennedy, M.D., professor of cardiology at Rush Medical College in Chicago, received the organization's highest honor, the Citation of Merit. The award is given annually to one alumnus in recognition of the physician's contributions to the medical profession. Kennedy, who graduated from the school in 1964, was chief of cardiology at St. Louis University Medical Center from 1982 to 1989.

The Outstanding Young Physician Awards, given to alumni under age 45 who have distinguished themselves in medicine, went to three graduates from the 1970s. **R. Paul Fairman, M.D.,** a 1972 graduate, is an associate professor of medicine and the director of the Medical Respiratory Intensive Care Unit at the Medical College of Virginia in Richmond. **Sheldon L. Kaplan, M.D.,** a St. Louis native who graduated from the school in 1973, is a professor of pediatrics at Baylor College of Medicine in Houston, TX. **Leslie W. Miller, M.D.,** who earned his medical degree in 1974, is an associate professor of medicine and surgery at the St. Louis University School of Medicine.

Two 1960s graduates received Service Awards, which honors alumni who have distinguished themselves through community service. **Michael S. Clarke, M.D.,** who earned his medical degree in 1969, practices orthopedic surgery in Springfield, MO. **Othniel Seiden, M.D.,** a 1964 graduate who retired from private general practice in 1978, is founder of Doctors to the World, an organization that provides health care for the needy in developing countries and in the United States.

The Medical Alumni Organization also honors individuals who are not MU School of Medicine alumni but have demonstrated a special interest in the school and the organization. This year four individuals, three of them MU faculty members, won Honorary Alumni Awards. **George W.N. Eggers, M.D.,** professor of the American Society of Anesthesiologists. **Ann Harvey, M.D.,** associate professor of pathology, has received numerous teaching awards and is known for her devotion to students. **Haskell Monroe, Ph.D.,** professor of history, was honored for his support of the school during his four and half years as MU chancellor. **Dorothy D. Reister, M.D.,** of Shawnee Mission, KS, is a pediatrician who currently is president of the Missouri State Medical Association. She recently was named Doctor of the Year by the Metropolitan Medical Society of Greater Kansas City.

WASHINGTON UNIVERSITY

Proliferative diabetic retinopathy and proliferative vireoretinopathy, may soon be treatable as a result of the identification of the amino acid chain that detaches the retina from its supporting membrane.

Thomas A. Ferguson, Ph.D., assistant professor of ophthalmology and visual sciences, led a team of investigators in examining the chemical mechanism in the two conditions.

The retina is said to detach when the photoreceptors become separated from a thin layer of cells known as the retinal pigment epithelium (RPE).

Proliferative diabetic retinopathy, which affects more than one million diabetics, involves the invasion of blood vessels into the vitreous humor.

Proliferative vitreoretinopathy occurs in five to ten percent of patients who have laser surgery to reattach the retina. It can also result from some types of serious eye injury.

Although the two conditions differ, the mechanism leading to retinal detachment is almost identical. Dr. Ferguson focused on a molecule called VLA-2, the receptor that the cells use to bind to the viterous humor and contract it. VLA-2 binds to a sequence of amino acids in collagen, the major structural component of the vitreous. The researchers conducted a series of experiments with VLA-2 showing that cultured RPE cells contract the vitreous humour using that receptor. The peptide sequence allowing the binding of VLA-2 with the gel in the retina has been identified as ADGEA.

Dr. Ferguson thinks that by inhibiting the process of vitreous gel contraction and somehow stopping the cells from contracting, it will be possible to prevent the retinal detachment problem. The key is to keeping the cells from contracting lies in preventing VLA-2 from binding to the eye's viterous humor, and the tool that allows the binding to take place is the peptide sequence ADGEA.

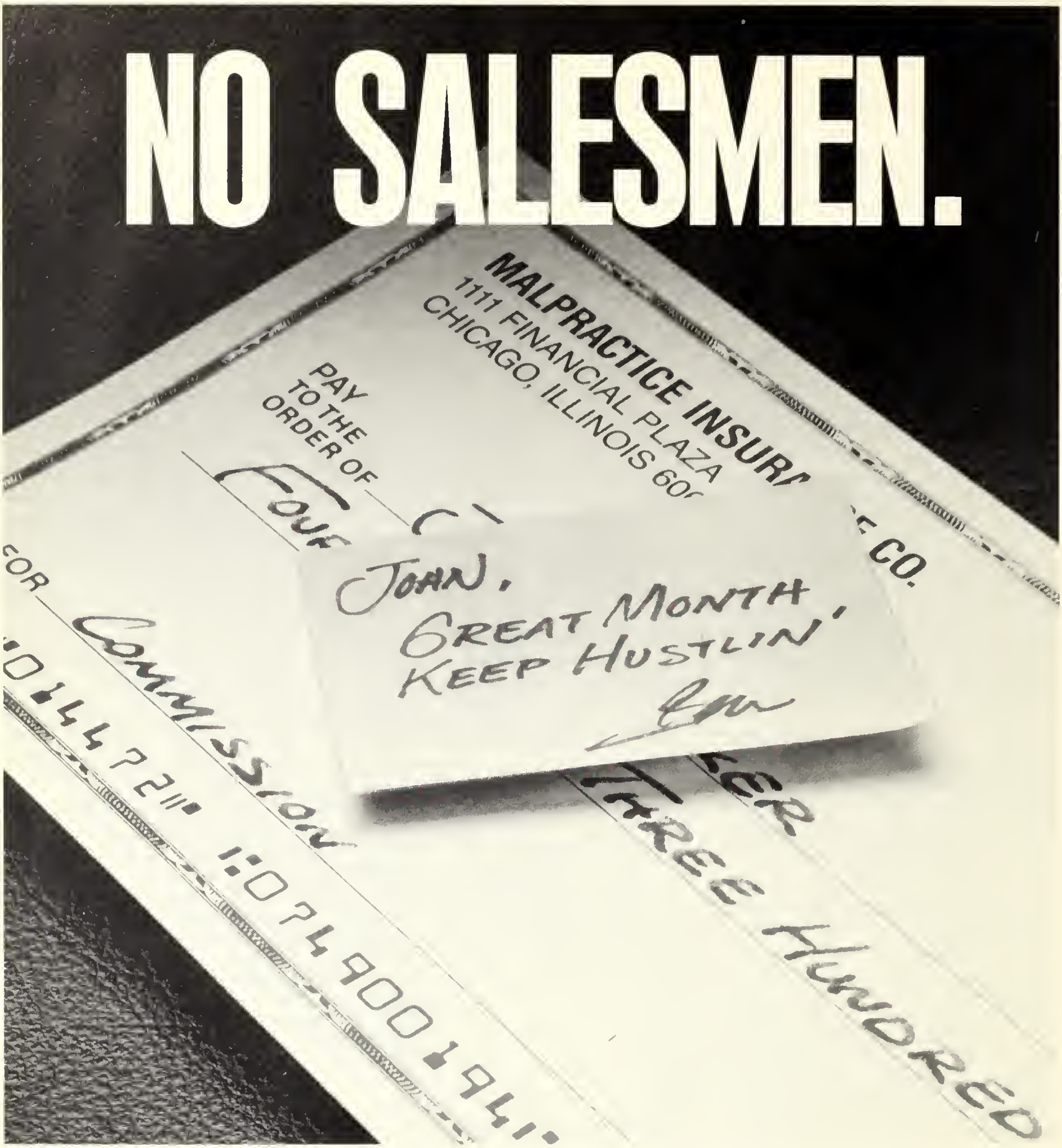
Jay S. Pepose, M.D., Ph.D., associate professor of ophthalmology and visual science, is studying ways to prevent reactivation of the herpes virus simplex virus in the eye.

In two studies, Dr. Pepose looked at factors that can induce viral reactivation in mice. Based on his research, Dr. Pepose suggests acyclovir be considered as a prophylactic treatment in patients with herpes infections of the cornea that reactivate frequently.

The drug would also be given to patients undergoing excimer laser surgery. This type of laser surgery. has the potential to reactivate the herpes virus.

In the second study, Dr. Pepose found that the excimer laser can reactivate the virus in mice. Between 20 and 60 percent of the mice in the study had the herpes virus reactivated. The reactivation rate depended upon how many laser pulses and how deep into the cornea the procedure probed. Yet, knowing that the laser can reactivate the virus and also knowing that oral acyclovir can prevent reactivation could lead to safer procedures. Many clinicians already use the drug as a matter of course. Dr. Pepose feels that producing this kind of data showing that oral acyclovir can prevent reactivation helps.

Many eye patients would have to take acyclovir constantly for it to have the desired prophylactic effect. While the side effects of taking this drug are few, it is costly. Frequency of reactivation will have to be weighed against the cost. At the very least, Dr. Pepose feels that patients who have excimer laser surgery on their eyes should receive the drug to prevent reactiviation of the virus that could defeat the purpose of performing the surgery.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June 1992

2 to 4 - *St. Louis*: Doppler Echocardiography- The Quantitative Approach. 20 credit hours. Contact Dianne Reid, St. Louis University Medical Center, 314/577-8897.

3 - *St. Louis*: Pulmonary Embolus in Thromembolic Disease. Contact Judith L. O'Guin, St. John's Mercy Medical Center, 615 S. New Ballas Rd., St. Louis, MO 63141, 314/569-6290.

5 - *Kansas City*: Prostrate Cancer. Contact The Ed Bixby Institute for Postgraduate Medical Education, 2316 E. Meyer Blvd., Kansas City, MO 64132, 816/276-4306 or 800/892-5771.

4 to 6 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part I: Peripheral Nerve. 22 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

4 to 7 - *St. Louis*: Advances in Aesthetic & Reconstructive Breast Surgery. Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 S. Euclid, St. Louis, MO 63110-1093. PH 800/325-9862, 314/362-6893.

6 - *Kansas City*: Fourth Annual Lipid Conference. Jointly sponsored with UMKC School of Medicine. 3 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

6 - *St. Louis*: New Concepts in the Management of Parkinson's Disease. 5 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

7 to 9 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part II: Spine-Microsurgery & Stabilization. 24 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

8 to 12 - *St. Louis*: Neuro CT Program. 25 credit hours. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

10 - *Kansas City*: Domestic Violence Awareness. 3 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

10 to 14 - *St. Louis*: Practical Anatomy & Surgical Tech. Workshop Part III: Microsurgery of the Brain. 36 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

11 to 13 - *St. Louis*: Cornea & Contact Lens Course. Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 South Euclid, St. Louis, MO 63110-1093. PH: 314/362-6893 or 800/325-9862.

12 to 13 - *Columbia, MO*: Common Childhood Problems - For the Practitioner Caring for Children. Contact Barry King, University of Missouri-Columbia School of Medicine, Office of CME, MA215 Health Sciences Center, Columbia, MO 65212. PH 314/822-4105.

15 to 17 - *Athens, Greece*: International Conference on Ischemic Syndromes. 16 credit hours.Contact Ralf Lucus, St. Louis University Medical Center, 314/577-8811.

15 to 19 - *St. Louis*: Body CT Program. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

16 to 18 - *St. Louis*: St. Louis Summer Geriatric Institute. 23 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

18 to 21 - *Tan-Tar-A, Osage Beach*: 44th Annual Scientific Assembly of Missouri Academy of Family Physicians. For more information contact MAFP, 8301 State Line Road, Suite 102, Kansas City, MO 64114, 816/363-0322 or 800/942-8347.

19 - 21 - *Lodge of the Four Seasons, Lake Ozark, MO* - **Annual Professional Meeting of the Missouri Chapter of American College of Surgeons.** Contact: Linda Wheelen, 1926 South Broadway, Springfield, MO 65807, 417/869-5222.

22 to 26 - *St. Louis*: Magnetic Resonance Imaging of Head & Body. Contact Louis A. Gilula, M.D., Mallinckrodt Institute of Radiology, 510 Kingshighway Blvd., St. Louis, MO 63110-1076, 314/362-2916.

25 to 27 - *St. Louis*: Pediatric Advanced Cardiac & Trauma Support & Pediatric Advanced Life Support Provider Course. 20 credit hours. Contact Mary Beth Foerstel, R.N., St. Louis University Medical Center, 314/577-5385.

Help for the Impaired Physician

Through its Physicians' Health Committee, MSMA helps doctors who are suffering from alcoholism, other drug addiction, psychiatric disorders, or senility. The thrust of the program is rehabilitative, not punitive. The Committee is composed of physicians who have special expertise in these areas, some from personal experience. Effective treatment for these illnesses is achieved most easily when the disease is detected early and family, friends, and associates are urged to avoid misguided sympathy which enables the condition to deteriorate.

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Send your check today to the Missouri State Medical Foundation, 113 Madison St., P.O. Box 1028, Jefferson City, MO 65101.

CME - Continued from page 377

26 to 27 - *St. Louis:* **Phacoemulsification Course: Transition and Intermediate Level.** Contact Surgical Care Foundation Education Center, 450 N. New Ballas Road, St. Louis, MO 63141, 314/991-2526.

26 to 28 - *St. Louis*: **Frontiers in Endourology: Laparoscopic Nephrectomy and Beyond.** (Urologists Only) Contact Cathy Caurso, Office of CME, Washington University School of Medicine, Box 8063, 660 South Euclid, St. Louis, MO 63110-1093. PH: 314/362-6893 or 800/325-9862.

July 1992

10 & 11 - *St. Louis*: **Advanced Trauma Life Support Course.** 16 credit hours.Contact Mary Keegan, R.N., St. Louis University Medical Center, 314/577-5385.

11 to 18 - *London, England & Lyon, France*: **Southern Association for Geriatric Medicine 2nd Annual Meeting.** For more information call the Southern Association for Geriatric Medicine, 800/423-4992 or 205/945-8425.

15 to 17 - *St. Louis*: **Pediatric Advanced Cardiac & Trauma Support & Pediatric Advanced Life Support Provider Course.** 20 credit hours. Contact Mary Beth Foerstel, R.N., St. Louis University Medical Center, 314/577-5385.

18 - *St. Louis*: **The Physician's Role in Smoking Cessation.** 4 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

28 & 29 - *St. Louis*: **Transesophageal Echocardiography Course**. Contact Dianne Reid, St. Louis University Medical Center, 314/577-8897.

31 to Aug. 1 - *St. Louis*: **Advanced Cardiac Life Support Provider/Recertification.** Course. 16 credit hours. Contact CME Office, St. Louis University Medical Center, 314/577-8167.

August 1992

4 & 5 - *St. Louis*: **Transesophageal Echocardiography Course.** 13 credit hours. Contact Dianne Reid, St. Louis University Medical Center, 314/577-8897.

5 to 7 - *Whistler, British Columbia*: **Southern Orthopaedic Association 9th Annual Meeting.** For more information contact the Southern Orthopaedic Association at 800/423-4992 or 205/945-1848

5 to 8 - *Kansas City*: **Management of Obstetric Emergencies.** 17 credit hours. Contact Carol Osborne, CME Office, St. Luke's Hospital of Kansas City, 816/932-3737.

13 to 15 - *Hilton Head Island, SC*: **Southern Association for Oncology 5th Annual Meeting.** For more information contact the Southern Association for Oncology at 800/423-4992 or 205/942-0530.

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Classified - continued from page 379

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In addition, in view of The Copyright Revision Act of 1976, effective Jan. 1, 1978, transmittal letters to the editor should contain the following language: **"In consideration of the Missouri State Medical Association's taking action in reviewing and editing my submission, the author(s) undersigned hereby transfers, assigns or otherwise conveys all copyright ownership to the MSMA in the event that such work is published by the MSMA."** We regret that transmittal letters not containing the foregoing language signed by all authors of the submission will necessitate delay in review of the manuscript. Please include the title of the article in the transmittal letter.

Manuscripts

Manuscripts should be typewritten, double-spaced, and the original with one copy submitted. Retain another copy for proofreading. Please number the pages of your manuscript. Manuscripts are not returned. Hospital and University appointments of the author(s) should accompany the manuscript. A synopsis-abstract of no more than 75 words should accompany the manuscript. Manuscripts may be submitted on 5 or 3 floppy disks using WordPerfect 5.1 or below.

References

References should be cited in numerical order in the text and arranged at the end of the article in the same order in which they are cited. The reference should give name of author, title of article, name of periodical, volume number, initial page number and year. Authors are responsible for reference accuracy.

Illustrations

Illustrations should be glossy prints or drawings in India ink on white paper. They should not be mounted and name of author and figure number should be penciled lightly on the back. Legends should appear on a separate sheet.

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Editorials should be written in clear, concise language. Length should be about two or three pages typed with double spacing. Letters will be published at the discretion of the editor, managing editor, and Editorial Board.

Deadlines

All material other than scientific should be received prior to the first of the month preceding month of publication.

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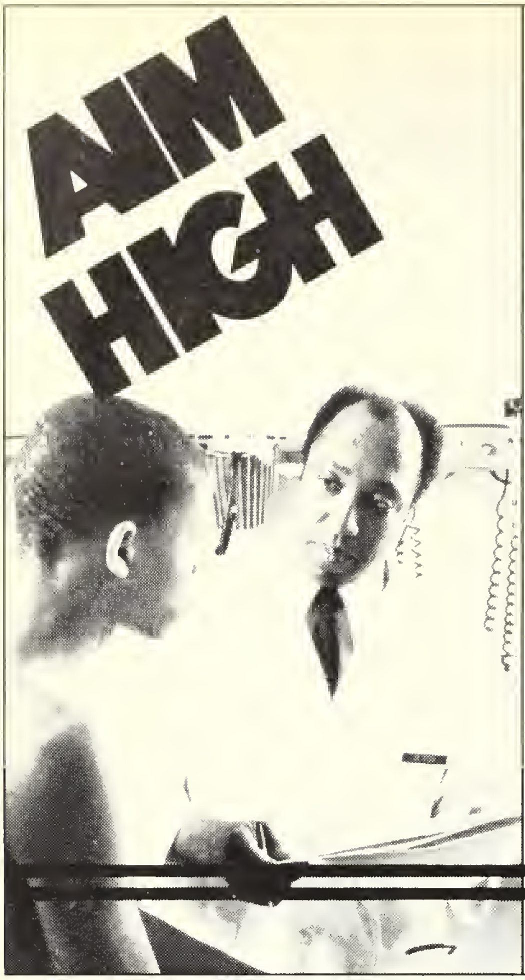

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Postage and Forms	☑	☐
Handle all mail	☑	☐
800# for inquiries	☑	☐
Electronic claims	☑	☐
Third party collection letters	☑	☐
Power Dialing	☑	☐
Refund overpayments	☑	☐
Multiple fee schedules	☑	☐
Software enhancements	☑	☐
Collection calls and letters	☑	☐
Unlimited training	☑	☐
Enter patient demographics	☑	☐
Generate HICA forms	☑	☐
Lock box deposits	☑	☐
VISA/Mastercard	☑	☐

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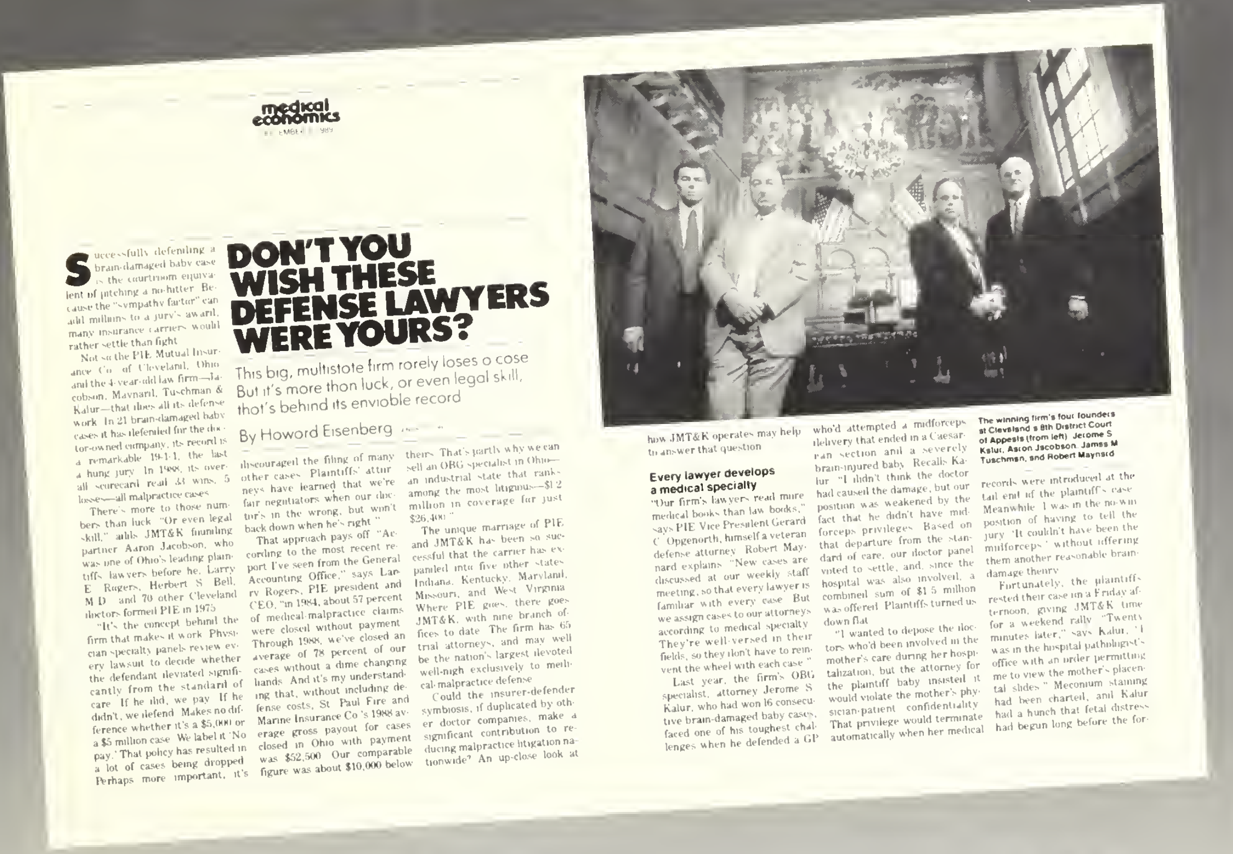

medical economics

DON'T YOU WISH THESE DEFENSE LAWYERS WERE YOURS?

This big, multistate firm rarely loses a case But it's more than luck, or even legal skill, that's behind its enviable record

By Howard Eisenberg

Successfully defending a brain-damaged baby case is the courtroom equivalent of pitching a no-hitter Because the "sympathy factor" can add millions to a jury's award, many insurance carriers would rather settle than fight

Not so the PIE Mutual Insurance Co of Cleveland, Ohio and the 4-year-old law firm—Jacobson, Maynard, Tuschman & Kalur—that does all its defense work In 21 brain-damaged baby cases it has defended for the doctor-owned company, its record is a remarkable 19-1-1, the last a hung jury In 1988, its overall scorecard read 33 wins, 5 losses—all malpractice cases

There's more to those numbers than luck "Or even legal skill," adds JMT&K founding partner Aaron Jacobson, who was one of Ohio's leading plaintiffs lawyers before he, Larry E Rogers, Herbert S Bell, M D and 70 other Cleveland doctors formed PIE in 1975

"It's the concept behind the firm that makes it work Physician specialty panels review every lawsuit to decide whether the defendant deviated significantly from the standard of care If he did, we pay If he didn't, we defend Makes no difference whether it's a $5,000 or a $5 million case We label it 'No pay.' That policy has resulted in a lot of cases being dropped Perhaps more important, it's discouraged the filing of many other cases Plaintiffs' attorneys have learned that we're fair negotiators when our doctor's in the wrong, but won't back down when he's right "

That approach pays off "According to the most recent report I've seen from the General Accounting Office," says Larry Rogers, PIE president and CEO, "in 1984, about 57 percent of medical-malpractice claims were closed without payment Through 1988, we've closed an average of 78 percent of our cases without a dime changing hands And it's my understanding that, without including defense costs, St Paul Fire and Marine Insurance Co 's 1988 average gross payout for cases closed in Ohio with payment was $52,500 Our comparable figure was about $10,000 below theirs That's partly why we can sell an OBG specialist in Ohio—an industrial state that ranks among the most litigious—$1 2 million in coverage for just $26,400 "

The unique marriage of PIE and JMT&K has been so successful that the carrier has expanded into five other states Indiana, Kentucky, Maryland, Missouri, and West Virginia Where PIE goes, there goes JMT&K, with nine branch offices to date The firm has 65 trial attorneys, and may well be the nation's largest devoted well-nigh exclusively to medical-malpractice defense

Could the insurer-defender symbiosis, if duplicated by other doctor companies, make a significant contribution to reducing malpractice litigation nationwide? An up-close look at how JMT&K operates may help to answer that question

The winning firm's four founders at Cleveland's 8th District Court of Appeals (from left) Jerome S Kalur, Aaron Jacobson, James M Tuschman, and Robert Maynard

Every lawyer develops a medical specialty

"Our firm's lawyers read more medical books than law books," says PIE Vice President Gerard C Opgenorth, himself a veteran defense attorney Robert Maynard explains "New cases are discussed at our weekly staff meeting, so that every lawyer is familiar with every case But we assign cases to our attorneys according to medical specialty They're well-versed in their fields, so they don't have to reinvent the wheel with each case "

Last year, the firm's OBG specialist, attorney Jerome S Kalur, who had won 16 consecutive brain-damaged baby cases, faced one of his toughest challenges when he defended a GP who'd attempted a midforceps delivery that ended in a Caesarean section and a severely brain-injured baby Recalls Kalur "I didn't think the doctor had caused the damage, but our position was weakened by the fact that he didn't have midforceps privileges Based on that departure from the standard of care, our doctor panel voted to settle, and, since the hospital was also involved, a combined sum of $1 5 million was offered Plaintiffs turned us down flat

"I wanted to depose the doctors who'd been involved in the mother's care during her hospitalization, but the attorney for the plaintiff baby insisted it would violate the mother's physician-patient confidentiality That privilege would terminate automatically when her medical records were introduced at the tail end of the plaintiff's case Meanwhile I was in the no-win position of having to tell the jury 'It couldn't have been the midforceps ' without offering them another reasonable brain-damage theory

Fortunately, the plaintiffs rested their case on a Friday afternoon, giving JMT&K time for a weekend rally "Twenty minutes later," says Kalur, "I was in the hospital pathologist's office with an order permitting me to view the mother's placental slides " Meconium staining had been charted, and Kalur had a hunch that fetal distress had begun long before the for-

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